BRAIN.
PARTS I. & II., 1893.

Original Articles.

ON DISTURBANCES OF SENSATION WITH ESPECIAL REFERENCE TO THE PAIN OF VISCERAL DISEASE.[1]

BY HENRY HEAD, M.A., M.D.
University College Hospital.

INTRODUCTORY.

SEVERAL years ago I was led to examine the positions occupied by pain in disorders of the stomach and I soon came to the conclusion that the usual description was incomplete in several respects. For firstly the positions over which the patient experienced pain in gastric disturbances were more numerous than was usually supposed, and secondly the pain was in many cases associated with definite cutaneous tenderness. Moreover the cutaneous tenderness was in many cases not confined to small spots or areas, but occupied whole tracts of skin with definite borders. I was thus led to investigate the pain and accompanying tenderness consequent on disturbances of other organs, and I found that these sensory disturbances also followed definite lines.

After Ross's most suggestive papers it seemed exceedingly probable that these areas bore some definite relation to nerve distribution, and I then began to investigate the distribution of herpes zoster in the hope that a skin lesion which was notoriously of nervous origin might throw some light on the meaning and significance of the tender areas in visceral

[1] Read as a Thesis before the University of Cambridge, June, 1892, and before the Neurological Society of London, November 10th, 1892.

disease. To my surprise I found that the areas occupied by herpes zoster exactly corresponded to those with which I was familiar in visceral disease.

I next attempted to determine to what level of the nervous system these areas belonged, with the help of cases in which gross organic lesions were present. By this means it became apparent that each of these areas represented the distribution of either a single nerve root or of a single segment in the spinal cord.

Thus I was enabled to map out the areas supplied by the various segments of the cord on the surface of the body. It then became apparent that certain of these areas were never affected in visceral disease, and this led me to examine the sensory supply of the viscera from the sympathetic system. Now Ross had already suggested that in visceral disturbances pain (and therefore in my cases tenderness) was referred along the distribution of the somatic nerves which came off from the same part of the cord as the sensory sympathetic fibres to the organ affected. Thus if I could map out the somatic areas along which pain was referred in visceral disease I could say, on Ross's hypothesis, what was the sensory supply from the sympathetic of the particular organ affected. By this means I obtained another scheme showing the distribution of the sensory sympathetic fibres analagous to that which Gaskell constructed for the motor and inhibitory fibres of the same system.

The next question that arose was whether these tender areas appeared only in visceral disease, and I soon found that they appeared to a greater or less extent in many general diseases, such as anæmia, ague and fever of all kinds.

An examination of the zones of analgesia or of tenderness in so-called hysteria showed that the sensory disturbances in this disease fall into two great groups—in one of which the areas of sensory disturbance followed the spinal type, whilst in the other the distribution was fundamentally different.

This opened up the whole question of sensation in its various forms, but in this paper I shall not do more than

touch upon the relations between the distribution of the sensation of pain, of heat, of cold and of touch.

In the present paper I shall confine myself to the discussion of the disturbances of sensation, &c., below the level of the clavicles, for although every general statement applies equally to the head and neck and the body below the clavicles, the problems opened up by a consideration of the areas of tenderness occurring in the head and neck are so wide reaching that I must reserve them for a subsequent paper in the near future.

CHAPTER I.

General.

§ 1. Characteristics of the areas.

§ 2. The relation between the pain areas and the distribution of Herpes Zoster.

§ 3. Topographical.

§ 1. *The pain and areas of tenderness in visceral disease; their characteristics, and the means for their demonstration.*

In order to appreciate the characteristics of the areas of tenderness which appear in visceral disease it is best to take an example. In the type of case which is so common in our hospitals where a patient suffers from pain after food with or without hæmatemesis, an area of cutaneous tenderness is sometimes found which can be marked out as follows. The skin and subcutaneous tissues are taken up gently between the finger and thumb at various points on the abdomen. As soon as we pass a line extending from a point about two and a-half inches above the umbilicus to the tip of the eighth rib the patient at once complains of pain. On working from below upwards, tenderness is complained of as soon as a line is passed extending from the umbilicus to the tip of the tenth rib. The tenderness may lie entirely to the left of the middle line of the abdomen or there may be an exactly similar area on the right side.

If unilateral the tenderness is in the form of a triangle with the base at the linea alba, one side formed by a line with an upward slope from the umbilicus to the ribs, and

the third side extending from the eighth rib to a point on the middle line of the abdomen two and a-half inches above the umbilicus. The apex is situated at about the ninth space. Now this anterior portion is associated with a posterior portion, also roughly a triangle. Its base is formed by the eleventh and twelfth dorsal spines, and its apex reaches a little further forward than the line of the posterior axillary fold.

A still better method of marking out such an area is to take a pin with a round head of such a size that it is obviously blunt to all parts of one's own cheeks and face. Travel over the surface of the abdomen using the blunt end only, in the same way as if you were using the point to test for analgesia. In a favourable case the patient does not complain until the limits of the tender areas are reached, when he at once complains that he is intensely sore and he will even cry out that you are pricking him. Thus in a favourable case the increased sensibility is so great that the contact of the blunt head of a pin is mistaken for a prick of the point. Subsequently in this paper this phenomenon will appear in my notes of cases as "head of pin called point." In a large number of cases there is no such confusion, but the patient says that, although he knows that the blunt end is being used, it gives him great pain. He feels "sore and bruised, as if he had been beaten about the body or back."

It is wonderful how unanimous patients are in their description of the sensation produced by touching such a tender area. I for one can fully confirm the description usually given by patients of their sensation; for such tender areas when touched, give exactly the sensation produced by touching a bad bruise. Many of my readers will have at one time or another suffered from these tender areas, and will be able to bear out my description.

I must warn my readers from supposing that the patient cannot tell the difference between the head and the point of a pin on these areas. Far otherwise. The touch of the head of a pin feels to him like a prick on a normal skin surface, whilst the point gives him acute pain far in excess of that caused by a prick upon the normal skin.

Another method for bringing out these tender areas, which is clumsy but theoretically interesting, is the use of a warm sponge or of a test tube filled with moderately warm water. As soon as the tender area is reached the patient experiences a feeling of great heat and flinches under the application. The same effect can be produced by moderate cold except that even moderate cold is at all times unpleasant to the skin of the abdomen, whilst moderate warmth is pleasant, and thus brings out the contrast between the normal and the altered sensory areas in a much clearer manner.

Another characteristic of these tender areas is the increased reflexes which can be obtained from them. This is a subject to which I shall return later, but I may mention here that whenever a portion of the skin becomes hyperalgesic from visceral disturbances the reflexes normally obtainable from that area are much increased. This is beautifully seen when the hyperalgesia affects the skin of the abdomen or buttock on one side only, so that the reflexes on the two sides can be compared with one another.

Immediately below the area of the abdomen which I have just described lies a tender zone which frequently appears in visceral disease. This area is bounded above by a line running from the umbilicus to the tip of the tenth rib and thence directly backwards to the first lumbar spine. Now supposing this sub-umbilical area is tender, no matter how intense the tenderness may be, its upper border never extends beyond this upper limit. Should the area which I have described above the umbilicus be marked out by tenderness its lower border never extends below the same line, however tender it may be. Again should the sensation of pain be entirely absent over either of these areas, in consequence of a destructive lesion to the spinal nerve roots or cord, we never find that the upper and lower limits of these two areas encroach upon one another *provided a prick is the test used for anæsthesia*. Thus it is evident that these areas do not overlap one another, and it is a peculiar characteristic of these areas of painful sensation that wherever they are situated on the surface of the body, whether they be marked

out by hyperalgesia or analgesia, their limits never materially alter or overlap. Thus if we take any three areas adjacent to one another, of which the uppermost and the lowest are tender, the clear space between them of unaffected skin will represent the full extent of the middle area. This is both curious and unexpected, for we know that the areas on the skin supplied by the nerve roots [1] overlap considerably, and as a necessary result a circumscribed lesion does not produce a sharp border of anæsthesia to touch.

I have so far spoken as if a visceral lesion always caused tenderness over the whole of its appropriate area. This is not by any means the case. In disturbances of the stomach, instead of a whole tender area such as I have described above, we frequently find two tender spots from an inch to an inch-and-a-half in diameter situated over the costal margin at about the tip of the ninth rib, together with a posterior spot generally situated near the eleventh and twelfth dorsal vertebræ, a little to one side of the middle line. Now, if the patient is asked where his pain is situated, he will point to one or both of these spots. Again, it is possible that he may suffer from pain only, without any tender spots on the skin, much less a whole band of tenderness reaching to the middle line in front. But even if cutaneous tenderness is absent, the pain is referred to the position of the maximum tenderness corresponding to the affected areas. Thus in cases of stone in the kidney, the maximum pain is situated at the tip of the twelfth rib, and at this point we can frequently demonstrate considerable cutaneous tenderness ; this point obviously lies in the subumbilical area which I have described above. Every such area of cutaneous tenderness has one or more maximum points, the position of which is exceedingly important, for it is to the situation of these maxima that the patient refers his pain. Thus in each case, given the position to which the patient refers his pain, I can say to what area the pain belongs. For I recognise in these points to which the pain is referred the maxima of an area whose limits I have occasionally found marked out

[1] Sherrington, *Proc. Roy. Soc.*, vol. 52.

completely by tenderness. So fixed are these maxima, that supposing a whole area is tender, we can confidently predict that as the disturbance subsides, the tenderness will become limited to certain points, and when the disturbance has subsided still more, pain only will be left referred to the same points as the maxima. When I come to speak of the limits and relations of these areas, I shall therefore lay stress on the position of the maxima in each area as I describe it, for these maxima give us the clue to the meaning of pain referred to various spots on the body.

§ 2. *The relation between the tender areas in visceral disease and the areas affected in herpes zoster.*

Before I proceed to describe the limits of the tender areas in visceral disease, I must digress for a moment to point out how I have been helped in this investigation by certain trophic skin lesions, especially herpes zoster.

A controversy has long raged as to the true significance of herpes zoster, for it is at once evident to anyone who closely examines a case of herpes that the distribution of the vesicles does not in many cases follow the distribution of any peripheral nerve. As each case appears in the out-patient room, an attempt is made to show that it follows the course of an intercostal nerve, but such attempts mostly break down, and it is assumed that more than one nerve is affected. In Germany it has long been recognised that the eruption does not follow the line of any one nerve, and an attempt has therefore been recently made to explain its distribution by supposing it depends on the vascular system. This attempt, however, has not been satisfactory.

Now it has always been taught that herpes zoster is probably an inflammation of the posterior roots,[1] and that this explains the want of harmony between its distribution and that of the peripheral nerves. It therefore struck me that, if this were so, a careful collection of the areas occupied by herpetic eruptions would be an excellent

[1] *Von Bärensprung Annalen d. Charité*, Bd. ix. 2, Bd. x. 1, Bd. xi. 2.

method of mapping out the long sought distribution of the posterior roots. I therefore proceeded to collect cases of herpetic eruptions, and was astonished to find that they agreed in an extraordinary way with the areas of tenderness I had observed in visceral disease, for not only did the actual areas occupied by the eruption agree with the areas of tenderness, but I found that if the herpetic areas were carefully marked and measured, they were never found to overlap. Moreover the advent of the eruptions is mostly preceded by pain and tenderness of the skin. In three cases in which I was fortunate enough to see the patient before the eruption was developed, I was able to mark out an area on the skin which exactly corresponded to that seen in visceral disease, and I was unable at the time to say whether this area was due to reference from visceral disturbances, or was the percursor of a herpetic eruption. Again the herpetic eruption always makes its first appearance at certain points, from which in most cases it spreads. But even if the eruption spreads until it occupies the whole area, these points of first appearance are always the most affected. Now these points correspond to the positions of pain and maximum tenderness in visceral disease. Thus the areas occupied by herpetic eruption correspond to those which become tender in visceral disturbances in three points.

1st. They have the same distribution.

2nd. They do not overlap.

3rd. They have the same maxima.

Thus in the herpetic eruption I had a means of actually seeing the areas I was investigating, marked out for me on the surface of the skin.[1]

[1] I feel inclined to think that herpes zoster may be etiologically divided into two groups.

1st. The eruption may be secondary to irritation of the spinal cord, &c., by Caries or other nervous diseases, such as tabes dorsalis, or may be due to some nerve irritant such as arsenic.

2nd. The type of herpes which represents an acute specific. The onset of the illness is preceeded by 5 to 7 days fever thirst, loss of appetite, &c. The illness may begin with a rigor, or in a child with vomiting. The temperature falls with the outbreak of the eruption. The position of the eruption may, in some cases, be determined by long continued irritation, such as chronic cough, chronic intestinal catarrh, &c., but in many cases no such predisposing cause can be traced.

The herpetic eruptions which I shall use to illustrate this paper, are selected from a collection of sixty-two cases, all of which lay below the eighth cervical segment. Each case was drawn by myself from at least three points of view, and from a quarter to half-life size.

I cannot now enter into the question as to how far other skin diseases throw light on the distribution of these areas. I have interpolated the above short explanation in order that I may refer to the distribution of herpetic eruptions when we are discussing in the next section the limits of the areas of tenderness in visceral disease.

§ 3. *Topography.*

In the previous sections, I fear I may have led my reader to suppose that it would be an easy task to map out the areas of tenderness in visceral disease for the whole body. This is, however, not the case in practice. The first difficulty that meets us, is the only partial appearance of the areas in the forms of maximum tender spots. Now for these maximum spots to be of value, it is necessary that the whole area to which they belong should have frequently come under the notice of the observer, and yet it is only in a small proportion of the cases that he has the chance of seeing the area in its full development.

Then again, it is rare for one area only to be tender. In most cases, several areas are present at once; however, we frequently find that of three areas adjacent to one another, 1 and 2 will be present on one occasion, 2 and 3 on the next, and thus the limits of the three areas can be judged by exclusion. In fact, those cases where one area only is tender occurs so seldom, that if we had to depend upon such cases, many hundreds would be necessary before one could be found to satisfy our requirements.

But this leads me to speak of another difficulty which is by far the most formidable if we depend only on the areas which are produced by visceral disease. There are certain areas on the body which are extremely rarely affected. If they should be tender, they mostly appear in combination,

so that we are unable to split them up into their constituent elements. Thus of five neighbouring areas, 1 and 2, 2 and 3, or 1, 2, and 3 may be present together; but if 4 and 5 appear, they are never present separately, but always in conjunction with one or more of the other areas.

It might be asked, how do we know that the tract of skin which we assume to be composed of several areas is not in reality a unity? The answer to this question will be given in full later when we speak of organic disease of the nervous system; for in nervous diseases, we find those tracts which we cannot differentiate by means of visceral disease split up into their constituent elements. Again, the distribution of herpes zoster helps us in a few cases to break up these compound tracts into their elements.

I shall now give the limits of each area with its characteristics in detail, and in every case I shall give:—

1st. The area of tenderness.

2nd. The maximum spots.

3rd. The position of the pain.

4th. The distribution of a case of herpes (when possible), which corresponds to the area described.

Though I give bony points as the guides to these areas, I must warn my readers that they are not absolute guides; for the only true guides to these skin areas are skin points, of which, unfortunately, there are very few. Thus the umbilicus is a much safer guide to skin areas than any bony point. Men, women, and children differ to such an extent from one another anatomically, that no actual measurement from, or dependence on bony points will give us the true nature of a skin area; and although its mutual relations render its nature obvious, we should go astray if we were guided by its relation to bony points.

Subscapulo Inframammary.

The first area which I shall consider runs the course seen in fig. 1. Its upper border starts behind at about the sixth dorsal spine, and runs forwards under the angle of the scapula to a point in front in about the fourth space. The

lower border starts from about the eighth spine, and swings forward to the lower part of the sternum just above the base of the ensiform cartilage. This area cuts the eighth rib at the angle of the scapula; the seventh rib at about the level of the posterior axillary fold; the sixth rib at the anterior part of the axilla; the fifth rib in the nipple line.

The maximum points of this area consist of a spot under the posterior border of the scapula at about the level of the seventh dorsal spine, and about one-and-a-half inches from the middle line of the back; in front there is a well marked spot about the fifth rib, an inch internal to the nipple line.

The pain corresponding to this area, is said to be "between the blade bones" behind, and "just under the breast," or if situated on the left side, "just under the heart."

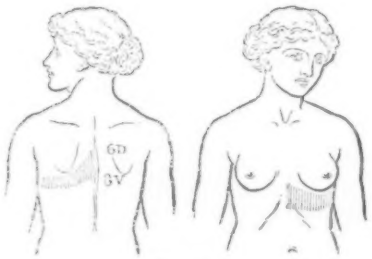


FIG. 1.

The shaded area represents the extent of the cutaneous tenderness in Case 1.

6D is placed at the level of the 6th dorsal vertebra. 8V at the level of the 8th dorsal spine.

Case 1.—Mrs. L. M., aged 35. Admitted Sept. 22. About two-and-a-half months pregnant. For rather over a month, constant vomiting. On admission to U. C. H., thin and wasted; constant vomiting and retching; pain under angle of scapula and under breast; cutaneous tenderness as in fig. 1. Sometimes right side, sometimes left side; sometimes more or less on both sides over this area; no tenderness elsewhere. Tongue furred; breath foul. No physical signs in heart or lungs. Pulse 96. Temp. 99.2. Urine normal. Erosion on cervix.

Twice much improved; relapsed again, and each time cutaneous tenderness reappeared. Ung. cocaine c. lanolin relieved pain and cutaneous tenderness, but did not stop vomiting.

October 23. Uterus cleared out.

November 9. Absolutely well. Has put on nearly a stone in weight.

A not uncommon form of herpes zoster is shown in the annexed fig. 2. It is never very marked over the spine, but a few spots are sometimes present over the sixth and seventh dorsal spines. There is always a well marked crop of vesicles over an area just internal to the angle of the scapula. In a well marked case the chain of vesicles run forwards, cutting the seventh, sixth, and fifth ribs, ending generally with a particularly well marked crop in the fifth space just internal to the nipple line. Sometimes two spots only are present corresponding very well with the maxima above mentioned. Thus we can see how closely this eruption corresponds with the area of tenderness. Moreover, in one of these cases, I was fortunate enough to be able to mark out an area of cutaneous tenderness of an intense character over this area before the herpetic eruption appeared.

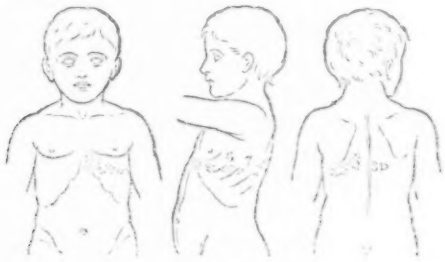


FIG. 2.

To show the distribution of the herpetic eruption in Case 2.

Case 2.—William S., aged 8. February 14.

Has complained for nearly two weeks that he could not raise his arm owing to pain in axilla.

Ten days ago, vomited. Went to bed with pain in the head and a sick feeling, but was well again next morning.

Four days ago eruption came out (fig. 2).

No surrounding tenderness.

Small glands in axilla.

Always has a slight cough and running at the nose. No physical signs.

Bowels open daily. No pain in stomach.

Here we have a typical case of zoster which runs round the body as a girdle crossing the line of several ribs, and not definitely following the course of one intercostal nerve.

The subscapulo inframammary area is characterised by

(1st) Two maxima, one just inside the angle of the scapula, the other over the fifth space under the nipple.

(2nd) Pain "between the shoulders" and "under the heart" on the front of the chest.

Subscapulo Ensiform.

This area consists roughly of two portions; a dorsal and a ventral triangle with their apices slightly separated from one another. It continually appears, especially on the left side, in stomach affections. [Fig. 27, p. 68.]

The base of the posterior portion is formed by the space between about the tip of the seventh or eighth spines and the nineth or tenth spines. Its upper border is formed by a line curving forwards about one and a half to two inches below the angle of the scapula to a point in about the seventh space at the level of the posterior axillary fold. The third side of the posterior portion is formed by an almost horizontal line from about the ninth spine forwards to meet the upper border just at the level of the posterior boundary of the axilla. The anterior half of this area is roughly a triangle, whose base is formed by a line extending from the base of the ensiform cartilage for from 2 to $2\frac{1}{2}$ inches along the linea alba. The upper border is a curved line from the base of the ensiform along the lower border of the sixth rib to a point in the sixth space, just behind the level of the anterior axillary fold. The third side of this anterior triangle is formed by a line extending from a point in the linea alba 2 to $2\frac{1}{2}$ inches below the base of the ensiform, directly outwards and backwards to meet the upper border in the axilla. Thus the apices of these two roughly triangular patches are separated from one another apparently by a distance which is a little greater than the breadth of the seventh rib.

The most common maximum point for this area is the

well-known "epigastric spot," exactly over the ensiform cartilage. The posterior maximum is situated close to the middle of the back, at the level of about the eight and ninth dorsal spine, and extending outwards to the line of the angle of the scapula.

The patient locates the pain corresponding to this area "over the pit of the stomach," *i.e.*, over the ensiform cartilage, and over the centre of the back at about the level of the eighth dorsal spine.

These are the well-known places to which pain is referred in so many cases of gastric ulcer.

I shall not spend much time in describing the herpetic eruption which corresponds to this area, for a glance at the rough figure I give of a case (fig. 3), shows how closely it resembles the area on fig. 27.

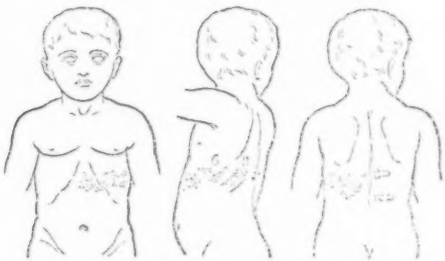


FIG. 3.

To show the distribution of the herpetic eruption in Case 3.

Case 3.—Harry P., aged 5, Nov. 9.

For two weeks has had a very bad cough. Feverish, hot and thirsty. No vomiting, except with the cough, which is so violent that he "heaves" with it.

November 4. During the night was very fidgety, could not sleep and complained of pain.

November 5. Spots began to come out. The patch in front came first. The posterior patch only came out yesterday.

Bowels regular.

Thus the subscapulo ensiform area is characterised by

(1st) Two maxima, one at the pit of the stomach, exactly over the ensiform cartilage, the other extending from the middle line of the back outwards to the angle of the scapula.

(2nd) Pain "at the pit of the stomach" and "just inside the shoulder blade."

Middle Epigastric.

This is a very puzzling area, and gave me a great deal of trouble before I could come to any conclusion with regard to its boundaries. It is particularly difficult to differentiate, owing to the fact that it is rarely marked out alone by tenderness in visceral disease. It most commonly appears with the one below it. It is, therefore, best made out by the method of exclusion, but I am fortunate enough to possess several cases where it appeared without its immediate neighbours. [*Vide* fig. 30, p. 74.]

The most important portion lies on the anterior surface of the body. Its upper limit consists of a line which starts from a point 2 to 2½ inches below the base of the xyphoid and passes upwards and backwards over the costal margin along the seventh rib. Thus far the upper border slopes upwards and backwards. But the remainder of the upper border of this area runs a different course. It starts from a point between the ninth and tenth spines, and runs horizontally out across the back, to a point about 2 to 3 inches from the middle line, just outside the line of the angle of the scapula. It almost touches the lower border of the subscapulo mammary (v. p. 74) area as it swings round the angle of the scapula. From this point the upper border rounds off downwards, just meeting the upper border of the anterior portions on the eighth rib.

The lower border behind is about one vertebra lower, if as much, and runs out parallel to the upper border until the angle of the scapula is reached, when it turns down to round off like the upper border.

The lower border of the anterior half of this area runs upwards and backwards from a point on the linea alba, about 2 to 2½ inches above the umbilicus, reaching the costal margin at the lower border of the eighth rib, and bending up to meet the upper border at about the middle of the axilla. Now fig. 30 shows very clearly the meaning of this curious conformation. For the blank spaces lying between the two

shaded areas exactly represents the tract occupied by the subscapulo ensiform area (p. 13).

However it is rare to find the area marked out with anything like this precision, for its dorsal portion is so insignificant that it is usual to find the anterior or abdominal portion well marked, but the posterior part only represented by the important maximum in the line of the angle of the scapula.

The more important maximum belonging to this area is the one situated about $1\frac{1}{2}$ to $2\frac{1}{2}$ inches below the level of the angle of the scapula and from 2 to 3 inches from the middle line. The second well marked maximum is situated in the eighth space, about 2 inches outside the nipple line and almost in the mid-axillary line.

The pain of this area is the well known "pain under the blade bone" behind and over the eighth space towards the anterior part of the axilla in front. Both points of reference are well marked in liver disorders, especially on the right side.

I give a figure (fig. 4) of a case of herpes zoster following this area very beautifully. The measurements were somewhat small, for the patient was only eleven years of age. This figure illustrates the difficulty of relying on absolute measurements, for the figure (fig. 4) obviously corresponds to the area in fig. 30, but all the measurements were slightly smaller.

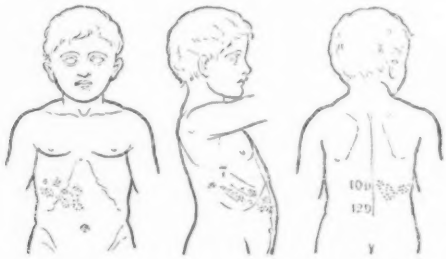


FIG. 4.

To show the distribution of the herpetic eruption in Case 4.
7 is placed over the 7th rib, and the sloping lines below it represent the 8th, 9th and 10th ribs.
10D represents the 10th dorsal vertebra.

Case 4.—Joseph S., aged 6, Oct. 26.

Has had a cold for a week, but eats well and is said not to have been "feverish."

Oct. 25th he showed the spots to his mother. She does not know which came out first.

No cough. Subject to bronchitis. Signs of emphysema with a few râles at the right base. Nothing definite in the lungs otherwise.

No vomiting. Bowels open daily.

Thus the middle epigastric area is characterised by

(1st) Two maxima, one in the 8th intercostal space, just external to the nipple line, and one about 2½ inches (in an adult) below the angle of the scapula.

(2nd) Pain "in the side," in about the 8th space, and pain "below the shoulder blade."

Supra-umbilical.

This area is simpler to describe than those which have preceded it. Its upper border extends from about the tenth or eleventh spine almost directly outwards across the base of the eleventh, tenth, ninth and eighth ribs. When nearly at the costal border it dips downwards to a point about 2½ inches above the umbilicus in the middle line. The lower border is formed by a line from the umbilicus upwards and backwards to the tip of the tenth rib; it then becomes a little more horizontal and runs almost straight back to a point corresponding about with the twelfth dorsal spine. [*Vide* fig. 26, p. 67.]

This area has a very well marked maximum point situated over the costal margin at the tip of the ninth rib (*i.e.* the tip of the tenth costal cartilage.) It lies partly on the costal border and partly just laps over to the abdomen and thus, when on the right side, exactly corresponds to the position of the gall bladder. The posterior maximum is not so prominent, but seems to lie at about the level of the eleventh dorsal spine about 1½ inches from the middle line of the back. Thus when double the patient complains of pain over the "middle of the back," and points to about the region of the eleventh spine over an area almost the size of the palm of the hand. In front the pain is always com-

plained of as "just on the edge of the ribs." When on the left side this pain "over the edge of the ribs" is a very common symptom of gastric disorder.

Herpes is not at all uncommon over this area. Fig. 5 shows a fairly well marked case in which the posterior portion is somewhat more marked than usual.

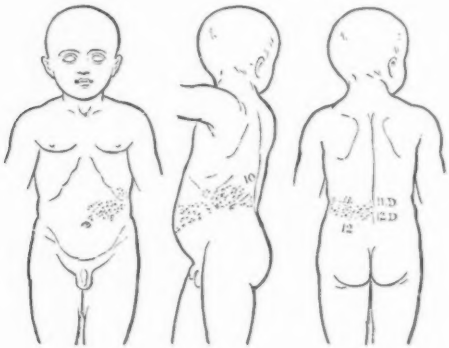


FIG. 5.

To show the distribution of the herpetic eruption in Case 5.
The eruption just reaches the umbilicus in front.
10 is placed over the 10th rib. The line above it represents the 9th, the line below it the 11th ribs.

Case 5.—Henry S., aged 3, Oct 28.

Very feverish, restless and irritable for a week.

Oct. 23, vomited, and again on Oct. 24. Since then his appetite has been very bad. Usually ravenous and can't be satisfied.

Bowels chronically out of order. Sometimes goes two or three days without a motion, and then many times in one day. Motions offensive. Rickety.

Cough. Bronchitis. A few râles over both bases. Rickety chest.

Oct. 28. Much better now the eruption has come out.

Thus the supra-umbilical area is characterised by

(1st) Two maxima, one over the tip of the 9th rib at the costal border and one over the back at the level of about the 11th dorsal spine, extending for a short distance outwards on the affected side.

(2nd) Pain "over the edge of the ribs," and over "the middle of the back," at about the level of the 11th and 12th dorsal spines.

Conclusions so far.

From what I have already said about these areas certain important conclusions can be drawn.

In the first place the areas of tenderness which appear as a consequence of visceral disturbance do not overlap. For if they overlapped to any considerable extent, like the areas of common sensation, the extent of skin covered when any one was present must necessarily be greater than that left unaffected when the areas on each side of it were tender. That is to say—supposing No. 1 and No. 3 were tender the skin between their borders, which was unaffected, must of necessity be of smaller extent than that affected when No. 2 only was tender. But this is not the case as is seen by comparing fig. 27 with fig. 30. The whole distance from the base of the xyphoid to the umbilicus is some 6 to 7 inches. Now of this distance the supra-umbilical area takes from $2\frac{1}{2}$ to 3 inches, the mid-epigastric $1\frac{1}{2}$ to 2 inches, and the subscapulo ensiform about $2\frac{1}{2}$ inches along the linea alba. If the supra-umbilical area only is affected there is found some $4\frac{1}{2}$ inches of unaffected skin between the base of the xyphoid and the upper border of the affected area; whilst if the two upper areas are affected the lower border of the affected tract of skin is found to lie some $2\frac{1}{2}$ inches above the umbilicus. Thus if the areas overlap they overlap to such a small extent as to be within the errors of clinical observation.

We are on more difficult ground when we examine the records of herpetic eruptions, for so many cases of herpes zoster occur in children and young adults that the absolute measurements give no clue to the position of the eruption. We are thus obliged to fall back on careful drawings not made on a plan, but with the whole figure drawn in proportion from nature and marked with the bony points and relative measurements. We can then say that although many cases of herpetic eruption do not occupy the whole

area we should expect, none of them transgress the lines I have already laid down, and absolutely none lie at an angle to or partly inside and partly outside the lines of the pain areas. This last point is particularly interesting, for most of the areas of visceral tenderness lie *across* the line of the ribs; in this they resemble the distribution of herpes zoster.

In order that this paper may not be too long I shall now describe the lower areas more in tabular form speaking only in full of those which present some difficulty.

Sub-umbilical.

This is an easy area to map out for it occurs more often alone than perhaps any other area in the body. [*Vide* fig. 28, p. 71, and fig. 34, p. 85.]

The upper border is a slightly sloping line passing round the body from the first lumbar spine to the umbilicus. The posterior border is formed by the first to the fourth lumbar spines, whilst the anterior border extends from the umbilicus to a point about 2½ to 3 inches below it in the middle line of the abdomen.

The lower border is very curious for it is not a horizontal line like the upper border, but shows a curious downward tag—the first indication of the profound alteration produced by the limb. The posterior portion of the lower border extends from the fourth lumbar spine directly forwards and downwards across the extreme upper part of the crest of the ilium to a point just posterior to the anterior superior spine. Here the lower border takes a sharp turn upwards and backwards as high as the level of the crest of the ilium. From this point it again goes directly forwards and downwards to the linea alba 3 inches below the umbilicus. Thus taking the sub-umbilical area altogether, it forms a broad band extending round the body between the lower ribs and the crest of the ilium with a curious tongue, which is so arranged as to miss the whole of the anterior superior spine of the ilium.

This area has two well marked maxima, one of which is

situated over the position usually occupied by the tip of the twelfth rib, the other over a point in front internal to the anterior superior spine of the ilium and about 1½ inches outside and 1 inch below the umbilicus.

The pain corresponding to this area is referred to the loins behind, over the tip of the twelfth rib and in front to a point in the iliac fossa somewhat external to and about on a level with the umbilicus. This is the area over which pain is referred in the cases of renal calculus. Patients suffering from this disease walk with the hand over the side affected, so placed that the thumb presses on the loins behind and the fingers on the iliac fossa in front.

Herpetic eruptions are common over this area and I have chosen a very beautiful one from my collection which is complete and shows the notch in a very perfect manner.

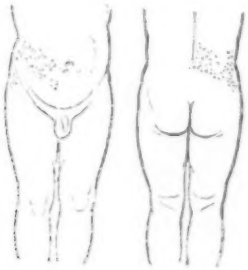


FIG. 6.

To show the area occupied by the herpetic eruption in Case No. 6.

Case 6.—William P., aged 4, Oct. 2.

No definite history could be obtained excepting that the eruption came out on Sept. 30. Bowels and appetite good. No cough.

Sacro-iliac.

This area is exceedingly difficult to obtain pure. Its maxima are constantly appearing in pelvic affections, but so soon as the whole area becomes tender some other neighbouring area is almost certain to appear more or less.

The case I have selected for fig. 35 is interesting in that on the left side, the area that we are about to consider is alone tender, whilst on the right side it is associated with the sub-umbilical area which I have just described. As will be seen from fig. 35 this area has almost lost its resemblance to a band owing to the disturbance produced by the limb. It may be divided into a posterior and an anterior portion. Posteriorly it reaches as high as the spine of the fifth lumbar and the descending flap on to the buttock occupies roughly the sciatic notch and extends about 2 inches below the level of the highest point of the crest posteriorly. Anteriorly this area forms a simple iliac and inguinal flap reaching the middle line of the abdomen about $2\frac{1}{2}$ inches above the pubes. It does not supply the part covered by the pubic hair in either male or female. There is also the tendency to the formation of a slight lateral flap. This cannot be seen on anterior or posterior diagrams, but requires a diagram of the side view of the body.

It is very interesting to notice that this area completes the one above, filling in the fork completely.

The two maxima belonging to this area are well marked and of frequent occurrence in pelvic disorders. Posteriorly we mostly find a spot of tenderness over the fifth lumbar and first sacral spines, extending outwards for at least 2 inches from the middle line of the back. In front the maximum is situated just above the position of the internal inguinal ring.

The pain corresponding to this area, is the "pain at the lower part of the back" (*i.e.*, over the fifth lumbar and first sacral spines), which so constantly occurs in women, especially during labour. It shoots round the hip "to the groin" (patient places his hand almost over the internal ring). If the pain is not sharp and shooting, it is fixed "in the lower part of the back and in the groin."

I have only once seen a nearly perfect herpetic eruption over this area [fig. 7], for most of them are more or less incomplete. Some are incomplete posteriorly; some anteriorly. I give a figure of the one which appeared to me to be most nearly complete of any I have yet seen.

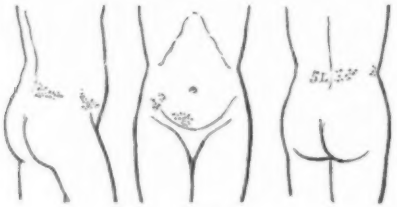


FIG. 7.

To show the area occupied by the herpetic eruption in Case No. 7.

Case 7.—Clara W., aged 12, December 21.

She has had pain over the lower part of her stomach for some time.

December 20. Eruption came out.

There is well marked cutaneous tenderness over a somewhat wider area than the extent of the eruption. This is so tender, that the head of a pin is called the point.

I also give the notes of an incomplete case [No. 8] for it was the case in which I first saw a complete area marked out by tenderness before more than a few vesicles had appeared. I was asked to see a case in the wards who was suffering from intense pain over the right side low down in the abdomen, and I discovered he had a very few herpetic vesicles. I could not then determine to what area they belonged, but mapped out the cutaneous tenderness, which followed very closely the limits of the area we are now discussing. The herpetic eruptions gradually developed and although it never became quite complete, it marked out the greater part of the area on fig. 7.

Case 8.—James H., aged 40. U. C. H. (Dr. Bastian).

Bronchitis and asthma for several years. Fresh attack, with swelling of the legs, five or six weeks ago.

On admission.—Marked emphysema; mucous râles all over the chest, with crackling râles at both bases. Tongue fairly clean. Spleen and liver not felt. No piles. Bowels usually regular.

Urine, normal.

Nothing cardiac.

November 25. Did not sleep owing to severe pain in right loin.

November 27. I found he had a band of intense cutaneous tenderness, marking out the sacro-iliac area on the right side. A few small herpetic vesicles have begun to appear on the abdomen.

November 28. Temperature rose for the first time to 100.6. Frontal headache. Pains in arms and legs. Shivering. Tongue furred. Appetite poor.

December 1. Herpes has developed fully.

Sacro-femoral.

This is one of the hardest areas in the body to deliminate, for it rarely, if ever, occurs alone, and its boundaries are so complicated, that even if it were present, it would take a long time to mark them out at all carefully.

It occurs most constantly with the sacro-inguinal area just described, and the case fig. 31 shows the two areas at the same time. Posteriorly, it causes an increase in the downward flap on to the buttock, so that this flap reaches quite 4 inches downwards from the level of the highest point of the crest of the ileum. In addition, there is a second great lateral flap extending to the top of the trochanter, and still further forwards it forms a triangle on the front of the thigh, reaching from 3 to 4 inches below Poupart's ligament. It seems curiously enough to supply a very small lateral flap on each side of the scrotum, the greater part of the pubic hair in the male, and a very small spot at the root of the penis.

There seem to be two maxima belonging to this area, one of which is situated just below the crest of the ilium, about 4 inches from the middle line of the back, and a second maximum which is always well marked over the outer border of Scarpa's triangle, just below Poupart's ligament.

The pain corresponding to this area is said to shoot from the upper part of the buttock, round the crest to the upper and inner parts of the thigh. Such is the account given by women after labour in whom this area appears as "after-pain." When the cause of the pain is continuous as in acute epididymitis, the pain is described as aching. [Compare fig. 32, p. 80.] This pain is situated about 3 to 4 inches below the level of the crest of the ilium behind, and in Scarpa's triangle, close to Poupart's ligament in front. One of my patients said it was as if someone had knocked him

"in the groin, and in the back," the pain was the dull aching pain of a bruise.

I have seen but two cases in which this area only was marked out by herpes zoster. As far as I have yet seen, it has nearly always appeared in conjunction with the area above it, excepting only when it was very incomplete. I give [fig. 8] one of the two cases in which this area appeared alone, and at the same time was fairly complete.

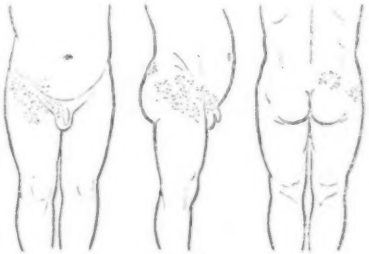


FIG. 8.

To show the area occupied by the herpetic eruption in Case No. 9.

Case 9.—William C., aged 5 (Dr. Crocker), December 23.

December 15. There was not a spot upon his body, and the child seemed perfectly well.

December 16. He was hot and feverish; wanting to drink water, but did not lie about or seem very ill.

December 18. The eruption began to appear as small blisters. No cough. No vomiting. Bowels daily. Quite normal. Appetite poor. Passes water well. No physical signs.

Gluteo-crural.

This area appears frequently in visceral disturbances but practically never alone. Its lower limits are easily defined for it is the last of this set of areas on the front of the thigh to appear in visceral disease. The case I have figured [fig. 36, p. 89], was one in which there was some catarrh of the cervix uteri. The cervix was dressed with copper and a Playfair's probe passed. As the result of the treatment she showed the tenderness I have figured, which passed off in a few hours. There was in addition a small spot corresponding to one of the sacral areas. The area figured was present

on both sides. This I take to be a case in which by a lucky chance this area above of all the lumbar areas was affected and it shows what I take to be its limits fairly well. It does not seem to reach the middle line behind but its most posterior point lies three inches from the middle line just at the level of the fork of the buttocks. This area shows to a greater extent than any we have yet seen the alterations produced by the limb; for here both the lateral and the anterior flaps are pulled out on to the limb to an enormous extent. It has two maxima; one especially well marked just above and to the inner side of the knee and one situated close to the great trochanter. The pain will be described more fully later on, and at present it will be sufficient to say that the great feature of the pain corresponding to this area is its shooting across the thigh to the inner side to end above the knee.

I have only once been fortunate enough to see this area marked out completely by herpes zoster (fig. 9). It mostly occurs either with one or two areas above it.

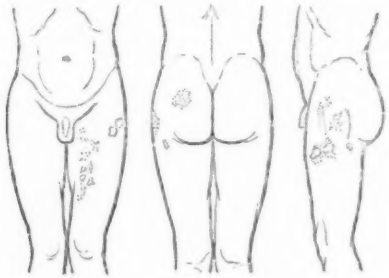


FIG. 9.

To show the distribution of the herpetic eruption in Case No. 10.

Case 10.—William E., aged 11, Oct. 16.

Eruption came out Oct. 2.

A week before this he suffered from headache and pain in the stomach. He shivered and complained that he was cold and had pains all over.

Then pain "at the side of his privates" came on and finally pain over the front of his thigh.

Weak, weedy looking child. Appetite bad. Bowels open every day. No cough. No physical signs in chest.

From this point downwards to the sole of the foot there are no areas which become tender in consequence of visceral disturbances excepting, as I shall show later, when some great change has taken place leading to universal tenderness of the whole leg. Thus from the visceral aspect the remaining skin in front of the thigh and leg is untracked desert. I shall therefore pass on to the posterior aspect of the thigh.

Sacral Areas.

The area of the skin now to be considered comprises the Perineum, Scrotum and Penis, a portion of the buttock and posterior aspect of the thigh as far out as the posterior border of the external hamstrings, the upper median portion of the calf and the sole of the foot. The areas which make up this tract of skin appear with decreasing frequency from above downwards, and if one of the lower ones is present, it is practically always accompanied by one of the areas above it. Thus I cannot give a case of visceral disease to shew each area separately but must show how the addition of each area alters the extent of the total skin affected.

Fig. 33 shows a tract of skin which is not uncommonly tender in painful affections of the mucous membrane of the bladder. The case from which the figure was obtained was that of a man from whose bladder a piece of mucous membrane was accidentally removed in the jaws of a lithotrite. In a second case an exactly similar area of tenderness appeared after the bladder had accidentally been washed out with 1 in 20 carbolic acid instead of with a harmless lotion. This tract of skin when bilateral is characterised by a small patch of tenderness over about the third or fourth sacral vertebra closely adjoining a saddle-shaped area on the back of the thigh; it extends into the perineum and covers the whole of the scrotum and penis excepting at the extreme root where it meets the sacro-inguinal area.

It will be noticed that I have coloured small portions of this tract differently in plates I. and II. I believe that the extent of skin shown in fig. 33 is in reality made up of two distinct visceral areas but as they almost always occur

together it is exceedingly difficult to separate them. However, I believe that in some cases of prostatic disease the area marked S 4 on plate IIA, extending forwards to a point just in front of the anus, was not affected. In a case of stone impacted in the ureter it certainly was not tender whilst the front part of the perineum was sore to the touch.

Thus I believe we have here to do with two areas one of which is so small that it may easily be missed and is situated over the sacrum reaching as high as a point corresponding to the second sacral spine. It passes forwards over the coccyx to the skin around the anus. It also in all probability takes in the inferior half (or even the whole) of the glans penis with a small portion of the inferior surface of the skin of the penis.

The remainder of the tract of skin figured (fig. 33) corresponds to a second area—the gluteo-pudendal, which occupies almost exactly the parts in contact with a chair in sitting. In addition it occupies the whole of the scrotum and the remainder of the penis.

In these two areas taken together there are three prominent maximum tender points, which constantly appear in disease of the pelvic viscera. The posterior one is situated over the lower part of the sacrum and possibly belongs to the sacro-anal area. Another very common tender point is situated almost exactly over the ischial tuberosity whilst a third is situated at the tip of the glans penis. Pain corresponding to these areas is said to come from the back and "shoot into the privates." The patient also complains of a feeling of weight and pain in the perineum and has an aching bruised feeling over the ischial tuberosities. He also complains of the pain over the glans penis so well known as a symptom of stone in the bladder.

I shall reserve a consideration of the areas mapped out by herpes until I have described all the areas of this group; for a herpetic eruption so seldom appears over one area only in this group that a preliminary knowledge of the distribution of this group is necessary for the due comprehension of the herpetic eruptions occurring in this region.

In profounder disturbances of the pelvic viscera we some-

times find an area marked out in the back of the thigh corresponding to the areas marked S 4, S 3, and S 2, on plate IIA. Now the whole of the upper portion of this tract obviously represents the sacro-anal and gluteo-pudendal areas. Subtracting these, a big strip remains down the back of the thigh about three inches in width, reaching to the upper part of the calf and extending outwards to the posterior border of the external hamstrings [marked S 2.] This area never appears alone in visceral disease.

It has a very well marked maximum point over the upper part of the calf of the leg, which is a prominent feature in many pelvic disorders; there is also a second maximum situated about the middle of the posterior aspect of the thigh.

The pain corresponding to this area is the pain in the calf, which is so troublesome in some cases of prostatitis. It is much increased by walking.

The next addition to this tract is easily defined, not because it appears alone, but because it is separated from the rest of its fellows. It covers the whole sole of the foot and invades the dorsum, as in plate IIA, S 1. A posterior flap runs up to a point on the calf about 5 inches above the level of the malleolar line.

The commonest tender point belonging to this area is situated on the sole of the foot about 1½ inches in front of the heel. There also seems to be a second tender point in the anterior part of the sole just on the outer side of the base of the great toe.

Both these points are the seat of pain belonging to this area. Patients also frequently complain of "aching in the soles of the feet," which prevents their standing for any length of time.

The last area of this group to be added to the tract of skin I have described occurs but seldom in visceral disease. It is situated on the outer side of the leg, and swings over to the dorsum of the foot. The whole coloured area in fig. 34, represents the extent of the tenderness in a case of prostatitis, probably caused by the passage of instruments for the first time. Now the area over the buttock is the gluteo-

pudendal, the area on the back of the thigh and calf is the sciatic, the area over the sole of the foot the soleal, and we have a fresh addition on the outer side of the leg, and the dorsum of the foot. This area is never in any case affected alone, and rarely without the sole of the foot being first affected.

The characteristic tender points of this area are situated over the dorsum of the foot and outer aspect of the leg. It is to these points that patients refer their pain when this area is affected.

From this point onwards we again come to the gap which, as I stated above, is never affected in visceral disease. Thus this gap is now seen to lie between the gluteo-crural area on the lumbar side and the fibulo-dorsal area of the sacral group.

Herpes Zoster over the Sacral Areas.

Unfortunately herpes zoster on the posterior aspect of the legs is rare, and when it appears over one area it is particularly liable to spread to one or more neighbouring areas. Von Bärensprung[1] figures one view of a case where the eruption almost exactly coincided with the two upper sacral areas, as we saw them in fig. 33. This case I reproduce in fig. 10.

FIG. 10.

To show the distribution of the herpetic eruption in Case No. 11. [Copied from von Bärensprung, "Charité Annalen," Bd. ix., Heft 2, p. 101.]

Case 11.—From von Bärensprung, p. 101. Anna Rahn, aged 19. Taken in June 9th, 1861, on account of soft sores and bubo. Menstruation regular until eight weeks ago. June 11th began to menstruate. Menstruation ceased June 13th. In the night of June 12th she felt a burning sensation in the neighbourhood of

[1] *Loc. cit.*, Bd. ix. 2, p. 101.

the left tuber ischii, and the next morning a group of clear vesicles appeared on this spot. The eruption gradually increased until it occupied the area in the figure. She also had a patch on the buttock extending down the upper and inner part of the thigh for a short distance. Another patch lay just over the fold of the buttocks on the lower part of the sacrum. The praeputium clitoridis was affected.

This eruption seems to correspond exactly with the upper two sacral areas. The next one which I figure belongs to the next two sacral areas—the sciatic and the soleal. Neither area is quite completely represented, but I think the eruption is wide spread enough for us to say that it represents very fairly the soleal and sciatic areas. It is interesting to note how the gluteo-pudendal area is spared.

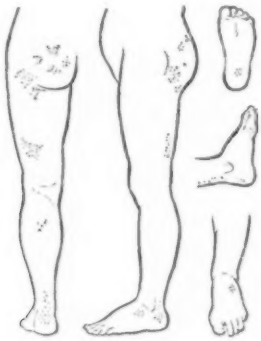


FIG. 11.

To show the distribution of the herpetic eruption in Case No. 12.

Case 12.—Harriet H., aged $5\frac{1}{2}$, October 11.

Two weeks ago complained of pain and aching in the feet. This aching lasted 2 to 3 days and then the eruption came out as a spot in the middle of the thigh. A day or two afterwards the gluteal spots appeared. The eruption on the foot was not seen until a week afterwards.

Appetite bad for months. Sick in the morning for a long while. Nothing in the perineum or anus. Bowels open daily, but sometime ago they were very irregular. No cough. Nothing in the chest.

The pain was very bad until 6 days ago, and the leg was very tender. No tenderness now.

The Upper Thoracic Region.

We have now followed these areas of tenderness from about the level of the fourth rib down to the thigh, and from the sacrum down the back of the leg. I had intended to describe the areas for the head and neck, and thus work downwards through a second gap, which we shall find in the arm, to the subscapulo inframammary area with which we started. A consideration of the problems opened up by a description of the areas on the head and neck would, however, make this communication of such inordinate length that I shall treat of the head and neck in a subsequent paper.

The lower limit of the lowest cervical area which is affected in visceral diseases is shown in plate I. and II. A and B. Now between this line and an area on the ulnar border of the fore-arm I have never yet seen tenderness as the result of visceral disease. Thus we here find a second gap, which can only be filled in later when we consider the disturbances of sensation produced by organic disease of the spinal cord and roots.

Dorso-ulnar.

Cardiac cases frequently complain of pain down the ulnar side of the forearm. This pain is occasionally accompanied by cutaneous tenderness. Fig. 21 shows this tenderness in a man with aortic disease, who frequently suffered from paroxysms of pain. After one of these paroxysms had passed off he was found to have intense cutaneous tenderness, as in fig. 21.

This area has at least three maxima, the posterior of which is situated over the first dorsal vertebra and a little to one side of the middle line, the anterior over the second space and third costo-sternal junction close to the middle line of the chest. A third maximum is situated on the inner side of the forearm, just below the bend of the elbow.

The pain over this area is of an agonising character, especially in cardiac disease. It is said to dart through from the back to the front and at the same time to appear

in the forearm, usually coming up from the little finger to the elbow.

I am able to give a most beautiful case of herpes exactly marking out this area.

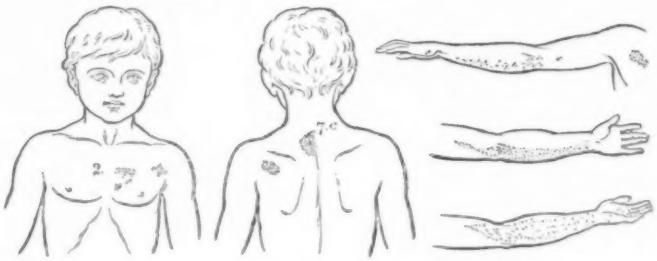


FIG. 12.

To show the distribution of the herpetic eruption in Case No. 13.
The figure 2 is placed over the lower border of the 2nd rib.
The figure 7c is at the level of the spine of the 7th cervical vertebra.

Case 13.—Henry George R., aged 7, August 17.

Eruption came out August 16. No antecedent pain. No cough. No physical signs. Appetite good. Bowels regular.

Dorso-brachial and Scapulo-brachial.

I can produce no case from my records in which each of these areas was separately marked out in its entirety. It is not at all uncommon in diseases of the chest to find the maxima of each area perfectly pure, but as soon as the whole of either area becomes tender it seems to bring the other with it.

It will be better therefore to consider the herpetic eruptions belonging to this group before proceeding to the tenderness produced by visceral disease. Fig. 13 represents a very complete specimen of herpes over the dorso-brachial area. The eruption started at about the level of the second dorsal spine and a very marked patch of considerable size lay between the spine of the scapula and the vertebral spines. The posterior and inner border of the arm was affected as far as the elbow. In front there were two patches in the third space extending just internal to the nipple line.

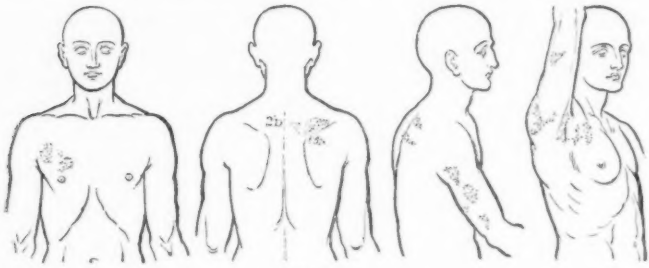


FIG. 13.

To show the distribution of the herpetic eruption in Case No. 14. Case No. 15 showed an almost exactly similar distribution.
2D is placed at the level of the spine of the 2nd dorsal vertebra.

Case 14.—James C., aged 27, carman, Feb. 11.

Feb. 10. Eruption first noticed. No antecedent pain excepting a slight feeling of soreness. In fact if he had not seen the eruption he would not have known that anything was the matter. Bowels quite regular. Appetite excellent. No cough. No physical signs. Never been ill in his life.

Very strong, healthy looking man with enormously developed muscles. Has been at work lately carrying very heavy wool-packs.

Case 15.—John R., aged 34, carman, Dec. 31.

For four or five days he had a feeling of weight over the third dorsal vertebra which made him constantly want to hold his head up. He frequently has this feeling in the back "whenever he has caught a little fresh cold." Having recently "caught a fresh cold" the sensation was not unexpected, but he was surprised to see the eruption come out on Dec. 30.

Eruption follows the same lines as Case 14, except that the arm is less affected.

Always has a cough, worst at night. No loss of flesh. Sibilant rhonchus at right apex in front, but at right apex behind distinct fine crepitations are audible.

Jan. 11. The herpes has gone. Considerable pain and tenderness over the second and third dorsal vertebræ. Also distinct tenderness over anterior part of axilla. Pain and cold feeling down the inner side of the arm. Fingers get bent up and numb, so that he cannot move them.

Note that he is a carman and has been engaged lately in carrying sacks.

Fig. 14 shows an eruption over what I take to be the third or scapulo-brachial area of the group. It reaches the middle line of the back about the level of the third dorsal spine. Its characteristic feature is the area over the subspinous

fossa of the scapula, which resembles a small scapula in shape. In front the eruption extends on to the extreme inner border of the arm for a short distance and a few spots appeared in the third space internal to the nipple line.

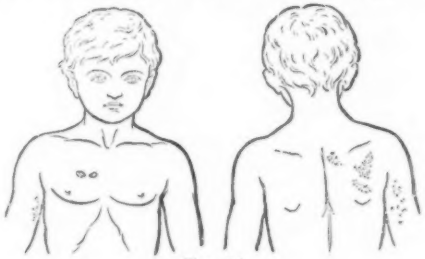


FIG. 14.

To show the distribution of the herpetic eruption in Case No. 16.

Case 16.—George C., aged 10½, Dec. 11.

Eruption came out Dec. 8. For three days previously he had pain over the area of the eruption.

Appetite poor for a week. Not feverish.

No cutaneous tenderness.

No cough. No physical signs.

Fig. 22 was obtained from a man who suffered from pain accompanied by cutaneous tenderness due to an aortic aneurism. The aneurism came forwards to the region of the second rib to such an extent that it was simply covered by skin. The area which it occupied is shown in the diagram (fig. 22A) and it was fortunately so situated that the referred tenderness did not correspond to the pulsating swelling. Now if we compare this figure with those showing the distribution of herpes (figs. 13 and 14) it is obvious that both areas are represented in consequence of the disturbance produced by the aneurism. But in addition a third area is present which I shall next describe.

The maximum tender points of the dorso-brachial or upper of these two areas are situated the one posteriorly exactly on a level with and over the more posterior point of the spine of the scapula and anteriorly in the third space exactly in and a little to the axillary side of the nipple line.

The characteristic tender point is situated posteriorly exactly over the centre of the scapula in the infraspinous fossa.

The pain of both these areas is said to pass through from back to front and to radiate down the arm, the one to the ulnar side of the elbow, the other to about the middle of the arm.

The Dorso-axillary.

This area is well shown, in plates I. and II., D 4. It is of frequent occurrence in lung disease.

The maximum tender points of this area are situated the one just internal to the posterior border of the scapula, the second over the posterior axillary fold and the third at the nipple, which always feels painful when this area is affected.

On fig. 15 we see another form of herpes occupying what I take to be the fourth area of this group. It starts from the middle line of the back at about the fourth dorsal spine, and swings outwards and downwards across the scapula. It also affects the posterior axillary fold, and goes into the axilla. In front, it forms a marked area above the nipple, sending a projection down towards the nipple itself, which is not, as a rule, affected in this type.

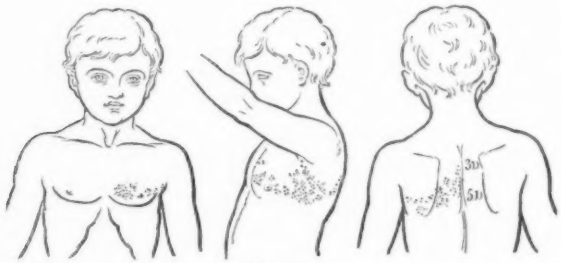


FIG. 15.

To show the distribution of the herpetic eruption in Case No. 17.
3D is placed at the level of the spine of the 3rd dorsal vertebra.
5D is placed at the level of the spine of the 5th dorsal vertebra.

Case 17.—Herbert D., aged 12, October 10.

He has been hot, feverish, and restless for three or four nights. Has a cough off and on for years. For the last 3 to 4 days the cough has been much more troublesome.

October 9. Eruption came out.

No pain. No tenderness.

Cough. No alteration in percussion note. Râles all down left side, and a few on the right base. Appetite poor. Bowels regular.

October 12. The eruption has developed, and now covers the whole area.

The Scapulo-axillary.

This area is frequently affected in all painful affections of the glandular tissue or ducts of the breast. Cracked nipples and over lactation will produce it, but it also occurs in men as I shall show later in consequence of malignant disease of the œsophagus. It is characterised by a dorsal patch close to the posterior border of the scapula at the level of about the fifth spine. It swings round the scapula to the axilla where it forms a marked flap, and ends in front in a patch just under the nipple. [*Vide* fig. 35.]

The maximum tender points are situated over the posterior patch, over the axillary patch, and over a point just on a level with, and internal to the nipple.

The pain is said to be "between the shoulder blades" and "just under the nipple."

A case of herpes belonging to this group is represented in fig. 16. It starts at about the fifth dorsal vertebra, and swings round the angle of the scapula all the vescicles lying above the border of the bone. In front, it is represented by a group of vescicles external to, and a group internal to the nipple, but the nipple itself remains quite free.

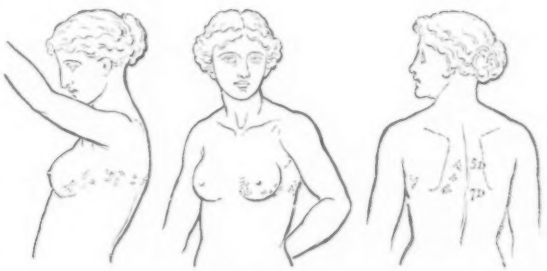


FIG. 16.

To show the distribution of the herpetic eruption in Case No. 18.
5D is placed at the level of the spine of the 5th dorsal vertebra; 7D at the level of the spine of the 7th dorsal vertebra.

Case 18.—Mary Ann L., aged 38, married, October 16.

October 1. Pain all up the left side. Shivering, cold, feverish. Loss of appetite. Bowels confined. Pain and giddiness in the head.

October 6. The eruption came out underneath the axilla. Pain continued till October 15, and she had great pain between the shoulders.

Has had eight children. Is now weak and run down from suckling a child of 17 months to prevent conception.

Now the next area below this is the one we started with, and is fully described on page 10.

Conclusions.

In this chapter I have described a series of areas which are tender in disturbances of the various organs. From the highest dorsal area (dorso-ulnar) I have traced 13 areas which fit into one another, and do not overlap to any appreciable extent. I have given cases of herpes zoster to show that the eruption in this disease appears over exactly the same areas as those which are tender in visceral disease.

We then passed to the buttock and the back of the leg, and marked out in a similar manner five areas belonging to the lumbo-sacral group. Between the lowest of the dorso-lumbar group, and the highest of the lumbo-sacral set is a gap which is not affected in visceral disease. This gap I shall fill in in the next chapter with the help of organic lesions of the nervous system, and a few cases of herpes zoster.

CHAPTER II.

The Significance of these Areas.

§ 1. To what level of the nervous system do they correspond?

§ 2. The numerical determination of the areas at their origin in the spinal cord.

§ 1. *To what level of the Nervous System do they Correspond?*

I have described the areas of tenderness occurring in visceral disease from the arm downwards and shall now attempt to determine the level of the nervous system to which they belong.

Now as far as visceral disease only is concerned these areas might correspond to a cortical or spinal distribution or

they might represent the supply of posterior roots or peripheral nerves.

It is exceedingly unlikely that they bear any relation to cortical distribution for as I pointed out above they are identical with the areas of herpetic eruptions and they closely correspond as I shall show below to the areas of loss of sensation in organic lesions of the spinal cord.

Thus these areas which are tender in visceral disease must belong primarily to the lower level of the nervous system and must correspond to the distribution of spinal segments, spinal nerve roots or peripheral nerves.

Any one who has attempted to describe the distribution of a herpetic eruption in terms of peripheral nerves knows how difficult it is and how seldom the results are satisfactory. Take for instance the area shown on fig. 2. It starts behind at the back of the seventh spine, crosses the ninth space at the angle of the scapula and in its course to the front cuts the eighth, seventh and sixth ribs and finally ends partly in the fifth space.

Again the twelfth dorsal nerve passes over the curve of the ileum and supplies a skin flap closely resembling the trochanteric flap of the sacro-femoral area [p. 24]. But this in no way resembles the analgesia in a case in which the twelfth dorsal nerve was cut in an operation on the kidney. The borders of the resulting loss of sensation were not sharply defined, but one gradually passed from complete to partial analgesia. Moreover the area of analgesia partly occupied the position of at least two pain areas and covered neither completely.

The improbability that these areas correspond to the distribution of peripheral nerves is best shown on the limbs. For though in the upper limb the patch on the inner side of the arm evidently represents the intercosto-humeral the patch on the ulnar side of the arm back and front (dorso-ulnar area) running down to the little finger represents no peripheral nerve.

Again the gluteo-crural area corresponds fairly well with the supply of the external cutaneous and the internal cutaneous nerves from the lumbar plexus. But the middle

cutaneous, which apparently is more ultimately associated with the internal cutaneous at its origin, is never affected.

Thus it is evident that these areas do not represent the supply of peripheral nerves.

Now if these areas do not represent a cortical distribution or the supply of peripheral nerves we have but two alternatives left. They must either correspond to the supply of spinal segments or to that of spinal nerve roots.

Ross held the view that the pain of visceral disease was referred along the line of nerve root distribution. But at the time his paper was written our knowledge of the supply of the nerve roots was more limited. Moreover he depended on the patient's account of his pain—a most unsatisfactory guide.

When I therefore discovered that the pain was associated with cutaneous tenderness it was at once evident that if Ross's ingenious hypothesis was correct I possessed in this tenderness a means of determining the supply of the various nerve roots by a physiological rather than by an anatomical method.

Now if we map out the distribution of the anterior primary division of any nerve root in the mid-dorsal region, and then add the distribution of the posterior primary division of the same root, the resulting tract will closely represent one of the areas I have described.

Moreover, von Bärensprung held that herpes zoster was an inflammation of the ganglion of the posterior root, and as we have repeatedly seen, there is a remarkable similarity between the areas occupied by a herpetic eruption and those which become tender in consequence of visceral disease.

Thus at first sight it appeared as if Ross's hypothesis were correct and that the areas I have marked out represented the distribution of the posterior roots.

But more than a year ago my friend Dr. Sherrington[1] communicated to me the results of his experiments on the distribution of the posterior roots, and pointed out that the areas of cutaneous supply from any two roots overlapped enormously. Thus on dividing one posterior root, Sher-

[1] *Proc. Roy. Soc.*, vol. 52.

rington obtained no loss of sensation to touch, and he was obliged to adopt the following ingenious device, in order to discover the skin supply of any one root. If he wished to investigate the supply of the seventh dorsal root, he divided all the posterior roots from the first to the sixth dorsal, and from the eighth dorsal to the first lumbar. Thus he had left one root intact, dividing five roots above and five below. He was then able to mark out an area of sensation, bounded above and below by tracts of skin over which sensation was absent. The areas which he obtained by this method in the monkey, bear a close relation, as far as their position is concerned, to those which I obtained according to my method; but whereas his areas overlapped greatly, the areas I had marked out did not overlap at all.

Thus we at once meet with a difficulty in assuming that the areas I have marked out represent the supply of posterior nerve roots. The solution might be found in the following hypothesis :—

Sherrington's areas represent the root supply for the sensation of touch, whilst my areas represent a similar supply for the fibres subserving pain, heat, cold and trophic disturbances in the skin. And thus we might assume that whereas the distribution of the fibres for the sensation of touch in the various posterior roots overlap considerably, those for pain, &c., do not overlap.

But Sherrington expressly states that he used touch and pain stimuli indifferently as a test for the presence or absence of sensation, and thus the areas he marked out represented in all probability the supply both of the touch and pain fibres of one posterior root. Moreover, I have on five occasions, been able to observe the results produced by the surgical division of a single posterior root in man. There is of course a certain element of doubt about such cases, in that the division of the root occurred during an operation for disease. However, they all show complete absence of anæsthesia to touch after division of one posterior root. In most of the cases the sensation of pain, heat and cold was also not materially disturbed, but in some there was very slight and uncertain dulling of painful sensation.

Again, I soon found that the borders of the analgesia were much less definite in those cases where there was reason to believe that the roots were mainly affected, than in those where the spinal cord itself was mainly implicated. Thus my clinical experience, so far, leads me to think that the areas I have marked out do not represent the supply of an actual posterior root and I think we must assume that whereas Sherrington's areas represent the true root supply, my areas correspond to the supply, not of roots, but of segments of the spinal cord from which the roots in part arise.

If we confine our attention solely to the phenomena produced by visceral disturbance, we are obviously only permitted to assume that my pain areas represent the supply of portions of the spinal cord. For we must suppose that a sensory disturbance passes up the nerve fibres from the viscera to set up changes in the activity of that segment of the spinal cord in which they terminate; and that these disturbances of activity are referred to that portion of the skin which is supplied by the affected segment. And when we turn our attention to the results produced by localised organic lesions of the spinal cord, we find that the affected areas closely correspond to those which appear in functional disturbances of the viscera.

But this is only true for the sensation of pain, and does not hold for the sensation of touch, even when we have reason to believe that the substance of the cord itself is affected. On testing the majority of such cases it soon became obvious that the anæsthesia to touch had a very indefinite border. One gradually passed from the area where a touch was not felt, to one where the patient was in doubt. Thus the upper limit of the anæsthesia was not a definite line. On the other hand the upper limit of the loss of painful sensation was frequently quite definite. Thus I can but suppose that a large number of records of "anæsthesia," in cases of spinal injury, are in reality records of the loss of painful sensation or analgesia. In some of the cases in Thorburn's book[1] it is definitely stated that the loss

[1] A Contribution to the "Surgery of the Spinal Cord," by Wm. Thorburn. London, 1889.

to touch was not so marked as loss to pain, and Allen Starr[1] in his most interesting paper, definitely calls attention, in several of the cases which were under his own care, to this want of agreement between the loss to touch and the loss to pain. In every case the loss to pain was more extensive and more definite than the loss to touch. Thus I think we may assume that in most reported cases of spinal injury, unless touch and pain are specially noted (as in Starr's cases) the description of the "anæsthesia" corresponds in reality to loss to painful stimuli.

Now an examination of a series of cases of injury to the spinal cord itself brings out the close relation between the areas of analgesia produced by such injuries and the areas which are tender in visceral disease. It also shows that the central connections of the fibres for pain and those for touch differ considerably, for the loss to pain is nearly always more definite and more extensive than the loss to touch.

To sum up the results arrived at in this section, we must conclude that there is a fundamental difference between the areas marked out on the surface of the body by Sherrington and by myself in that, whilst his areas overlap considerably, mine do not overlap to any appreciable extent. Now whilst his areas undoubtedly represent the cutaneous distribution of posterior nerve roots, my areas, at least as far as they concern the tenderness produced by visceral disease, equally undoubtedly represent the cutaneous supply of segments of the spinal cord from which these roots in part arise. But examination of the records of traumatic lesions of the spinal cord shows that the disturbance of touch is less profound than the disturbance of the sensation of pain, heat and cold. Therefore the mechanism for the sensation of touch in the various spinal segments must overlap, whilst that for the sensation of pain, heat and cold does not overlap at any rate to the same extent.

Thus, in conclusion, we may, I think, assume that the areas which are tender in consequence of visceral disturbance and the areas occupied by the eruption in herpes zoster

[1] *Amer. Jour. of Medical Science*, July, 1892, and also "Familiar Forms of Nervous Disease." New York, 1891.

represent the supply of the fibres for the sensation of pain, heat and cold, and of the trophic fibres to the skin from various spinal segments. They also roughly represent the regional supply of posterior roots; but probably the difference between the cord supply and the root supply will be found to lie in the absence of overlapping in the cord and the existence of overlapping in the root supply at various levels.

§ 2. *The Numerical Determination of the Areas at their Origin in the Spinal Cord.*

If the areas which I have described correspond to the cutaneous supply of the nerves for pain in each spinal segment we shall be able to assign to each area its proper number. But both in the arm and in the leg we are checked by a gap if we depend only on the areas of tenderness in visceral disease. Thus it is impossible to begin at the fourth cervical root and count downwards or at the last sacral area and count upwards.

Fortunately however, we are able to learn the origin of a few of the areas from complete destructive lesions (traumatic) of the cord or of the roots. Thorburn gives an exceedingly instructive case of a lesion of the cord at the level of the first dorsal root. The intrinsic muscles of the hands, which we know are supplied from the first dorsal root, were paralysed. The anæsthesia was limited "with the greatest accuracy" to the side of the arm and fore-arm being bounded in front by a line running vertically from the front of the deltoid opposite the second rib down the centre of the triceps and forearm to the middle of the wrist; similarly behind the boundary was a vertical line down the centre of the triceps and back of the forearm to the centre of the wrist. In the hand the limiting line sloped both in front and behind from the centre of the wrist to the cleft between the fourth and fifth fingers, the little finger being completely anæsthetic, and others not so. The trunk was anæsthetic as high as the level of the second rib in front and the spine of the seventh cervical vertebra behind. Compare the upper line with that

in plate I. and II., or with the upper line found by placing together the three herpetic eruptions shown in fig. 12, 13 and 14, and they coincide in a remarkable manner. Now the words limited "with the greatest accuracy" makes me think that the limit given for the "anæsthesia" was in reality the limit of analgesia. For so accurate an observer as Thorburn would never have described the vague line of anæsthesia to touch as "accurately limited." Moreover I cannot but think that if touch, pain, and heat and cold have all been tested the fact would have been mentioned.

Thus I think we can assume that the area marked out on plate I. and II. by D 1 represents a disturbance in the course of the first dorsal. Having determined that the area marked D 1 on plate I. and II. represents the supply of the segment of the spinal cord corresponding to the first dorsal root we can now count downwards, and number each area as has been done on the coloured plates.

How far does this enumeration correspond to what we know of other cases of lesion of the cord.

On p. 69 Thorburn gives a case of fracture dislocation of the sixth dorsal vertebra. The anæsthesia extended on the trunk "as high as the eighth rib." This description corresponds very fairly with the upper limit of the subscapulo ensiform pain area [plate I. and II., D 7.] The cord was completely cut across at the level of the sixth dorsal vertebra. Now the sixth dorsal root arises from the cord at the lower border of the fifth dorsal vertebra and the seventh root was the highest whose origin was completely destroyed. From our calculation we concluded that the subscapulo-ensiform must be supplied from the segment of the cord corresponding to the seventh dorsal root.

Again fig. 17 shows the upper limit of analgesia, *i.e.*, loss of appreciation of the prick of a pin in a traumatic spinal case. Mr. Horsley operated and found that owing to a fracture of the first and second lumbar vertebræ the roots at the level of the body of the twelfth dorsal vertebra were firmly matted up and apparently destroyed. The cord itself was apparently uninjured and pulsated down to the end. Moreover the area supplied by the lower sacral roots was

unaffected. The reaction to faradism was completely lost in the muscles of the legs, calf, and back of thigh. Thus we must assume that the roots involved in this tissue were degenerated. The highest root which comes off at the level of the twelfth dorsal vertebra is the first lumbar. Now the upper limit of the analgesia corresponds very fairly with the lower limit of the sacro-femoral area [*vide* plate I. and II., D 12], which by enumeration from the first dorsal was assumed to correspond to the distribution of the twelfth dorsal root segment.

FIG. 17.

To show the disturbance of sensation in Case No. 19.

The left half of the figure shows the areas over which the sensation of touch was lost. Note the ill-defined edges.

The right half shows the areas over which the sensation of pain, heat and cold were lost. The edges are better defined, but are not absolutely sharp, probably owing to the fact that the loss of sensation was due more to affection of the roots than of the spinal cord.

Case 19.—James S., aged 33, fireman. U. C. H. (Mr. Horsley), November, 1891.

On night of April 14, 1891, went to bed drunk and walked out of the window 40 ft. from the ground. Was unconscious for three to four weeks.

Condition on admission.—Marked kyphosis in dorso-lumbar

region. Whole dorsal region arched and spine rigid. Summit of kyphotic curve formed by the exceedingly prominent spine of the twelfth dorsal vertebra which stands out fully three-quarters of an inch beyond the rest. This prominent spine has a bifid extremity and feels almost as if it were two spines side by side. No tenderness on firm pressure.

Lower limbs rigid and nearly motionless. The feet are in the position of talipes equino-varus. Feet and ankle joints are stiff. In the knee joint passive flexion is possible up to 30° without undue force.

Complete loss of power in all the muscles of the right lower limb. No independent action in psoas and iliacus, but he is able to raise his thigh from the bed by lifting up the pelvis. On the left side he can voluntarily contract the following muscles, rectus, vastus externus, and to a less extent vastus internus, abductor muscles including adductor magnus. No action in hamstrings or glutei on either side.

Muscles on front of thigh and tibialis anticus react on both sides to faradism. On the left side the hamstrings, calf muscles and flexor brevis digitorum react, but on the right side these muscles do not react to faradism.

Muscles on front of thigh and tibialis anticus react to galvanism on both sides. The muscles of the calf and hamstrings also react on the left side but not on the right. A stronger current is required on the right side than on his left. A. C. C. appears early but does not preceed K. C. C.

Sensation of touch lost over the areas in fig. 17 left half. Penis and scrotum unaffected.

Sensation of pain lost over the areas in fig. 17 right half. Penis and scrotum unaffected.

Sensation of heat and cold follows the same lines as those of pain (fig. 17 right half.)

Knee-jerk abolished both sides. Right plantar reflex abolished but left is present. Cremasteric reflex abolished on both sides. No ankle clonus. Abdominal reflexes normal. No affection of upper limbs, trunk or head. Discs and fundus oculi normal.

Urine dribbles away. Ammoniacal and contains much mucus. He suffered from cystitis for several months before admission. Has control over the rectum and sphincter ani. Bowels open every two or three days.

December 12, 1891.—Mr. Horsley operated. He removed the arches of the eleventh and twelfth dorsal, and first and second lumbar vertebra. Across the lamina of the second lumbar on the right side was a groove formed by a fracture. Lamina itself was tilted so that its upper edge was directed forwards as well as upwards in a plane about 45° to the frontal plane. Opposite the eleventh dorsal vertebræ the cord was normal and pulsated with respiration. Opposite the twelfth dorsal the theca vertebralis was much thickened of a yellowish white colour and pulsation was just visible. Opposite first and second lumbar colour and general

appearance was normal and pulsation was distinct. Over the twelfth dorsal vertebra the adhesion of the theca to the bone was so close that it was impossible to pass a probe between the theca and the anterior part of the vertebra. Theca was opened. Opposite the twelfth dorsal vertebra it was adherent to the posterior surface of the cord and also to the nerve roots. They were so thickly matted that it was quite impossible to disengage them. Made an uninterrupted recovery from the operation.

Therefore I think we can say that the area which runs down the ulnar side of the arm corresponds to the supply of the first dorsal segment, whilst the area which forms a flap on the outer side of the thigh and crosses the thigh to reach the inner side above the knee corresponds to that of the first lumbar root.

But now we reach the gap and can count no further from above downwards.

Let us then proceed to the consideration of the areas on the back of the thigh on the sacral side of this gap. The tract of skin figured on page 82, which was affected in consequence of a lesion to the mucous membrane of the bladder closely resembles the area of analgesia in a case of destruction of the spinal cord at the level of the third and fourth sacral roots reported by Oppenheim.[1] Now I pointed out above that I believe this area is made up of two portions, and I think we can now assume that the saddle-shaped area on the buttock, with its maximum tender point at the ischial tuberosity represents the supply of pain fibres from the segment corresponding to the third sacral root, whilst the one above it, with a well marked tender point over the lower sacral spines, corresponds to the fourth sacral.

If this be so, the long thin area on the back of the thigh will represent the supply of the second sacral segment, whilst the soleal area will correspond to the distribution of the first sacral segment. Thus if the spinal cord at the level of the first, second, third and fourth sacral roots were destroyed, we should expect to get loss of sensation to pain over an area made up of these four tracts.

Now Herter and Starr[2] report a case in which this lesion was present, and the resulting analgesia bears a fairly close

[1] *Archiv. f. Psychiat.*, xx. 298. [2] *N. Y. Med. Journal*, Aug. 9th.

resemblance to a combination of the four areas marked out by tenderness in visceral disease.

Now the next area above the soleal which appears in visceral disease occupies the outer side of the leg and swings downwards to cover the dorsum of the foot as far forwards as an inch from the root of the toes. Here it borders on the soleal area. If, therefore, our enumeration is correct, this tract of skin must represent the supply of the pain fibres of the fifth lumbar segment.

Here, again, we come to the gap or tract of skin which is not affected in visceral disease. Thus this gap is now seen to be bounded above by the lower border of the first lumbar area and below by the upper border of the region supplied by the segment of the cord corresponding to the fifth lumbar root. Here, then, our areas of tenderness are no help in the delineation of the roots.

Fig. 18 shows the extent to which pain, heat and cold were lost in a case under the care of Dr. Hughlings Jackson, which he kindly allows me to mention. The patient was a man aged 27, who nearly two years before admission to the National Hospital, Queen Square, had fallen from a scaffold. In the right leg we find that the long patch so frequently seen in lesions about this region, extending down the inner side of the leg to the root of the metatarsal of the great toe is unaffected; whilst on the left side it is analgesic. Now Starr associates this patch with the third lumbar root. My material at present is too scanty to decide this point, but I have so frequently found that this area is affected when the areas about the knee are unaffected, and *vice versa*, that I feel inclined to think that it forms a separate root area of the form I have shown in fig. 20. It extends from the anterior border of the tibia inwards and backwards to the middle of the calf. Downwards it reaches the base of the metatarsal bone of the great toe and above it is forked, to receive a downward process of the area above. Now as this area comes next above that already described as the fifth lumbar, it probably represents the distribution of the pain fibres of the fourth lumbar root.

FIG. 18.

To show the disturbance of sensation in Case No. 20.
The left half of the figure shows the loss of sensation to touch.
The right half of the figure shows the loss of sensation to pain, heat and cold.
Note that the loss on the right half is more extensive and better defined than that on the left half of the figure.

Case 20.—Edwin C., aged 27, labourer. Queen Square (Dr. Hughlings Jackson), May, 1892.

August 8, 1890.—Fell from a scaffold. Was not at first unconscious, but became so afterwards.

Condition on admission.—Intelligent, healthy-looking man. Over lower end of sacrum and coccyx is a large irregular scar, with a small superficial area of ulceration. This is a bedsore, which has evidently at one time been much larger than it is now.

His motion, sensation and reflexes above the level of the pelvis are normal. Cranial nerves and special senses normal.

Can flex right hip well. Flexion of left hip present, but feeble. Extension of both hips feeble, but better on right than on left side.

Can extend right knee well. Extension of left knee considerably less than right. Cannot flex either knee when lying on his back. When lying on his face he can raise the right foot off the bed and flex the right knee to a right angle. The left foot can only just be lifted off the bed in this position. This action appears to be due almost entirely to the semi-membranosus.

Absolute paralysis of both feet, and all the toes. Both feet, especially the left, are in a position of simple talipes equinus without contracture. No rigidity.

Considerable wasting of both legs below the knees, and of the flexor and extensor aspect of the thighs. All the wasted muscles are flabby and soft.

Electrical reactions.—

	Faradism.		*Galvanism.*	
	Right.	Left.	Right.	Left.
Quadriceps	good	strong current	A.C.C > K.C.C.	0
Hamstrings	0	0	0	0
Glutei	?	?	0	0
Tibial Group	0	0	0	0
Peroneal Group	0	0	0	0
Sural Group	0	0	0	0

Tactile Sensation.—Lost over the areas in fig. 18 left half. The sensation of the penis and scrotum are perfect.

Painful Sensation.—Lost over the areas in fig. 18 right half. Complete loss of painful sensation in penis and scrotum, excepting over a small area at the base of the scrotum.

Sensation of heat and cold accurately follow the lines of painful sensation (fig. 18 right half).

Localises well over the areas where sensation is present. No allocheiria.

Reflexes.— Knee-jerk slight on right side. Absent on left side. No ankle clonus. Plantar reflexes not obtained. Abdominal reflexes and all upper reflexes, well obtained.

He knows when he wants to pass water, but has no power of retaining it. He can feel the passage of urine when it reaches the orifice. Urine 1024, faintly acid, trace of albumen. No sugar.

Feels when his bowels require to act, but is not conscious of the actual passage of fæces. Has no power of retarding action.

Complete loss of sexual desire since the accident. No involuntary erections. Occasional emissions without erections.

The next area above this is extremely difficult to describe in words, and its limits are better grasped from an examination of fig. 20. A case of herpes zoster shown in fig. 19 occupies this area in addition to the areas which we have already learnt to associate with the fourth and fifth lumbar. The characteristics of this area are its long upward flap on the inner side of the thigh over the adductors, and its distribution to the area around and over the patella. It has a small downward flap on the inner side of the leg behind the knee which fits into the fork of the area below (fourth lumbar).

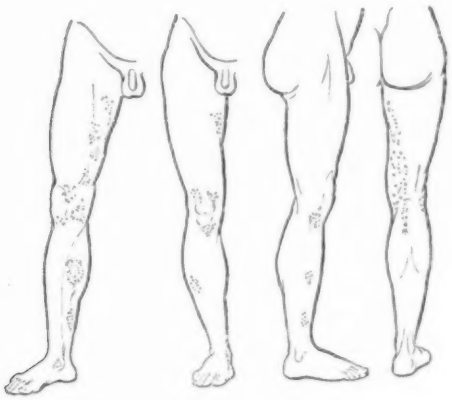


FIG. 19.

To show the distribution of the herpetic eruption in Case No. 21.
It is supposed to occupy mainly the areas belonging to the 3rd and 4th lumbar segments with slighter implication of the 5th lumbar.

Case 21.—Henry M., aged 22, June 28.

Last week he had a severe cold. He had a sore throat and a "whistling" cough.

Eruption came out June 19. No antecedent pain.

No tenderness except over the areas of the eruption.

Appetite poor. Bowels open regularly. No gonorrhœa or syphilis.

Thorburn (p. 132) gives a case of spinal injury where the upper limit of the anæsthesia was two inches above the knee joint. It was "concave downwards," and lower on the inner than on the outer side of the knees. This anæsthesia was caused by transverse divisions of the cord at the level of the origin of either the third or the fourth lumbar roots. As he expressly mentions that the posterior aspect of the leg was not examined when the anæsthesia was thus limited it is possible that this is the knee portion of the area I have just described.

This complicated area represents so far as I have yet seen the distribution of the pain nerves from that segment of the spinal cord corresponding to the third lumbar root.

Now if we place together on a figure all the areas we have determined so far, in the gap between the first and fifth lumbar roots, one only remains, having the following distribution. Its most posterior portion is situated over the buttock and does not quite reach the middle line behind.

It fills in the diamond shaped gap between the posterior part of the first lumbar and the upper part of the third and fourth sacral areas. Thence it sends a downward flap, whose posterior border corresponds to about a

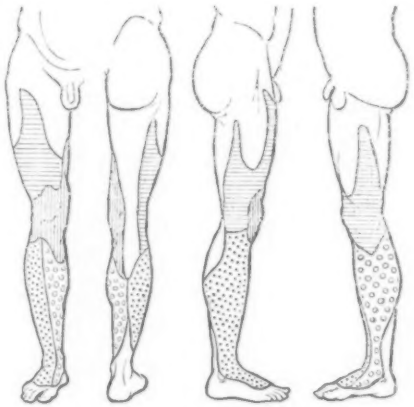


FIG. 20.

To show the presumed distribution of the areas supplied from the 2nd to the 5th lumbar segments. Of these the 2nd, 3rd and 4th lumbar comprise the "gap" which is not normally affected in disturbances of the the viscera.
The area marked in *cross lines* represents L 2.
The area marked in *vertical lines* represents L 3.
The area marked with *circles* represents L 4.
The area marked with *dots* represents L 5.

line drawn from the posterior border of the trochanter to the inner edge of the biceps at the knee. The anterior border of this flap is bounded by the posterior border of the descending flap of the first lumbar root area. On the front of the thigh it occupies the vacant space over the quadriceps, between the inner and the outer flap of the

first lumbar area and ends just above the knee, and on the inner side by adjoining the ascending limit of the third lumbar.

Thus we have now filled up the gap in the leg which is unaffected in visceral disease, and seen that it probably represents the supply of pain fibres from three segments, the second, third and fourth lumbar.

It would take me too long to discuss here how the gap on the arm is to be filled up, and I must reserve the full discussion for a future paper on the areas of the head and neck.

Now the descending branches of the cervical plexus which came off as low as the fourth cervical root supply the skin over the first two ribs and over the point of the shoulder. This represents the lowest limit of the areas of referred tenderness in the head and neck.

Thus the gap in the arm which is unaffected by visceral disease probably represents the distribution of the pain fibres of the fifth, sixth, seventh and eighth cervical segments.

Conclusions.

We have now been able, with the help of traumatic lesions of the spinal cord, to name each area according to the spinal segment to which it belongs. Thus the dorso-ulnar area represents the supply of the first dorsal segment, and the gluteo-crural that of the first lumbar, and each of the eleven intervening areas represents the supply of one segment whose number we now know.

In a similar manner we found that the gluteo-pudendal area represented the supply of the third sacral segment—the fibulo-dorsal that of the fifth lumbar, and the intervening areas have been numbered according to the segments to which they belong

We have also seen that the "gap" corresponds to the second, third and fourth lumbar segments, and we have been able from the lesions produced by organic disease to approximately apportion the skin into areas corresponding to each of these segments.

CHAPTER III.

Pain and Tenderness in Disturbances of Special Organs.

Introductory.

We have now seen that the whole of the body and limbs can be marked out into areas, each of which I have attempted to show, corresponds to the cutaneous distribution of the pain fibres given off from one segment of the cord. I shall now try and put before you the extent to which these areas are affected in diseases of the various organs.

It is a well-known fact that the pain of visceral disease is not always localised by the patient over the organ affected. Ross first explained this fact by the ingenious hypothesis that the pain of visceral disease was referred along the distribution of the somatic roots which came off from the same level of the cord as the sympathetic fibres to the organs affected. He collected a large number of cases of pain, and his intimate knowledge of the sensory distribution of certain roots enabled him to say definitely that in certain cases, the distribution of the pain corresponded exactly with that of certain roots.

The description given by patients of their pain is, however, so unsatisfactory, that this important work did not bear fruit to the extent that might have been expected.

An enormous advance was made when Dr. Mackenzie[1] described the cutaneous tenderness which is so frequently associated with the pain of visceral disease. For here we have a symptom which is distinctly more objective than that of pain only. He proceeded to collect a large number of cases in which the tenderness was present over one or more points, and in the course of this section, I shall repeatedly refer to his interesting paper. Although we worked independently and published at the same time, our results agree to an extent that is very flattering to me. In particular, I wish to emphasise the following points in which we are at one.

[1] *Medical Chronicle*, August, 1892.

1. The pain of which the patient complains always lies within the areas of tenderness, if these be present. Should they be present at one time and absent at another, but the pain remain, tenderness is always found to make its appearance in the area which is said to be painful.

2. The tenderness is not deep seated, but is purely cutaneous or subcutaneous. One of the best methods of eliciting it, is to pick up the skin gently between the finger and thumb. In addition, Mackenzie uses the point of a pin, but I generally use the broad blunt head of a large pin, especially where the area of skin affected lies over a bony substratum. Thus Mackenzie looks for the exaggerated expression of pain produced by the point of the pin over the tender area: whilst my method of stimulation produces no pain until the tender area is reached.

3. These areas of tenderness bear a definite relation to the different organs affected, but in many cases lie at a considerable distance from the organ affected. The pain produced by stimulation is not produced by any action on the organ itself, which may lie at a considerable distance from the tender spot. Again, the tenderness may be on the right side, though the organ affected lies on the left side of the body.

4. The superficial reflexes are usually exaggerated over the tender areas produced by visceral disease.

I shall now enumerate very shortly the areas which are affected in diseases of the various organs, and shall now use the segmental nomenclature I determined in the last section instead of the cumbrous names for the areas which were necessary before we had determined their significance.

The Heart and Aorta.

In diseases of the heart and great vessels, it is easy to get an account of the pain, but it is often difficult to demonstrate tenderness; for notoriously the most painful disturbances take the form of anginal attacks, in which the patient is not in a fit state to give an account of himself. Sometimes, as is well known, the pain radiates down the ulnar side of the

arm to the wrist or little finger, and sometimes it starts from this point and radiates up to the chest.

The first case I have selected for illustration, is that of a man, aged 40, who had suffered from attacks of pain three to four times a week. The heart was dilated and hypertrophied, and there was a double aortic and faint mitral systolic murmur. He first of all feels as if he were blown up with wind, then pain comes on under the left breast, and at the angle of the left scapula. It then appears at a point

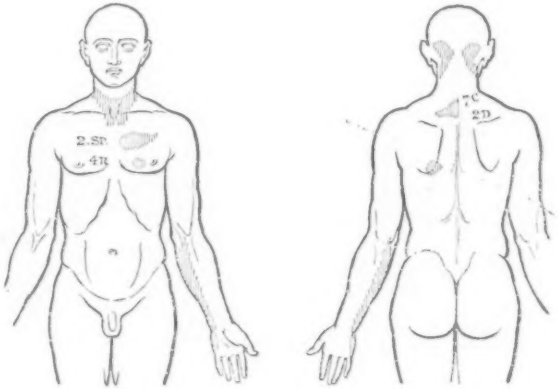


FIG. 21.

To show the areas of cutaneous tenderness during an anginal attack in a man with aortic disease (Case No. 22). They represent the whole of the 1st dorsal and 3rd cervical segments together with the maxima of the 4th dorsal.
2 Sp. is placed over the 2nd intercostal space.
4 R is placed over the 4th rib.
7 C at the level of the spine of the 7th cervical vertebra.
2 D at the level of the 2nd dorsal spine.

in the third space, just internal to the nipple line. In these attacks, a spot in the second space 3 inches from the middle line, and thence down the arm to the fingers, but not to the thumb. In the arm it darts backwards and forwards, sometimes upwards, sometimes downwards. I subsequently saw him in one of these attacks, and he then had intense cutaneous tenderness over the ulnar side of the forearm, back and front, over an area in the supra-spinous fossa at the level of

the second dorsal vertebra, and over an area in the second space extending from the nipple line to the mid-line of the chest. Thus the tenderness marked out very closely the area supplied by the first dorsal segment. In addition, he had pain and tenderness over the angle of the scapula, and over a point on to the fourth rib just internal to the nipple.

Case 22.—J. C., aged 40, draper's porter. Was in University College Hospital a few months before I saw him first with heart disease. The heart is now dilated and hypertrophied. Cardiac impulse strong. No thrill. Double aortic and mitral systolic murmur. Arterial pulsation up to ear. Capillary pulsation marked. Pulse 84, typically aortic regurgitant.

Suffers from attacks of pain as often sometimes as three and four in the week. He first feels blown up with wind, then pain comes on under left breast, and at angle of scapula. It then goes to a point just above the nipple; thence to a point in the second space from which it radiates down the arm to the hand. The ulnar side of the arm only is affected. After the pain in the arm comes on, he gets pain in the throat, with hoarseness and difficulty in speaking. In about an hour he vomits and brings up wind, and the attack is at an end.

On June 22, he had cutaneous tenderness (1) over the fifth rib internal to the nipple on left side. (2) Over the vertebral border of the spine of the scapula. (3) Over a point just internal to the angle of the scapula. (4) Over the ulnar side of the bend of the forearm. (5) Over the base of the hypothenar eminence in the left hand.

June 29. Came to U. C. H. in evening with a bad anginal attack. Attack began at 10.30 p.m. as described above. At midnight he was in great pain, face anxious and pale; great cardiac distress. Pulse could not be counted; was running and of very low tension.

There was intense cutaneous tenderness as in fig. 21, and the pain was referred entirely to the regions over which the skin was tender.

Voice hoarse. No cough. Pupils equal, about 5 mm. React normally.

He was put to bed and soon began to vomit. By 1 a.m., June 30, the attack was over.

At 8 a.m., June 30, he was well, and all cutaneous tenderness had practically gone. He left the Hospital to go to his work.

In the case of a man, aged fifty, who suffered from an aortic aneurism pointing to the region of the second rib, the tenderness was situated over the area shown in fig. 22, corresponding to the second, third, and fourth dorsal segments.

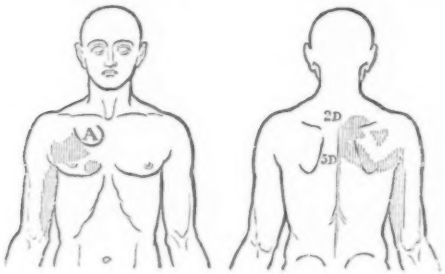


FIG. 22.

To show the areas of cutaneous tenderness in a man with aortic aneurism (Case 23). They represent the 2nd, 3rd and 4th dorsal segmental areas.
The outlined area A represents the extent of visible pulsation.
2 D is placed at the level of the spine of the 2nd dorsal vertebra.
5 D at the level of the spine of the 5th dorsal vertebra.

Case 23.—Edward T., aged 50, caretaker. U. C. H. [Dr. Roberts.]

November, 1890. Attack of gout. Pain in right side of chest and shortness of breath. By June, 1891, this pain had so increased that he could not walk without becoming short of breath and suffering from pain. July 15, U. C. H. Double aortic murmur, aortic pulse, aneurism of ascending and transverse portions of aorta diagnosed.

June, 1892, re-admitted. A little to right of middle line in first and second spaces is a swelling extending from the lower border of the clavicle to the third rib below. To the left it reaches the middle line of sternum; to right, nipple line. Surface of the pulsating swelling fairly regular except that there is a nipple-like projection in the centre.

Apex beat, fifth space half-inch outside nipple line. Diastolic aortic and less marked systolic aortic murmur over the swelling and over sternum.

Pulse 76, aortic. Pupils equal. No anginal attacks.

Pain on right side of chest:—

(1) in third space about 2½ inches from mid-sternum.

(2) Pain at extreme apex of axilla.

(3) And over a point behind at the level of the spine of the scapula a little to right of middle line.

July 3. Whilst singing hymns sudden increase of the pain and extension of the area of the aneurism; very marked cutaneous tenderness as in fig. 22.

Another case of dilatation of the aorta with dilated and hypertrophied heart showed the second dorsal segmental area in a very beautiful way. The maximum pain was situated

over the most posterior portion of the spine of the scapula and over the second space in front. This pain radiated down the arm, but did not go further than the elbow.

Mackenzie[1] gives a number of cases of cardiac disease showing pain and tenderness over these areas, and gives an excellent figure showing the whole area affected in such cases. He points out that in a large number of cases certain tender points make their appearance. A glance at plates I. and II. shows the segments to which they belong, according to my enumeration, but I will group these points according to the segmental areas of which they are the expression.

First Dorsal.— (*a*) Seventh cervical to second dorsal spine.
(*b*) Second space from nipple line to mid-sternum.
(*c*) Ulnar side of bend of elbow.

Second Dorsal.—(*a*) Over the spine and vertebral border of the scapula.
(*b*) Over the axillary border of the pectoralis major.
(*c*) Over the middle of the triceps muscle in the arm.

Third Dorsal.— (*a*) Over the middle of the scapula just below the spine.
(*b*) Over the third rib internal to the nipple line.

Fourth Dorsal.—(*a*) Over the third and fourth vertebral spines and outwards to the vertebral border of the scapula.
(*b*) Over the region in the axilla covered by hair.
(*c*) Just above the nipple itself.

Fifth Dorsal.— (*a*) Exactly over the angle of the scapula.
(*b*) Over the fourth rib and space internal to the nipple.

In cases of angina pectoris, whether there are physical signs of valvular disease or not, we frequently find that the

[1] *Loc. cit.*, fig. 4.

areas corresponding to the sixth and seventh dorsal roots are also affected. But in addition other areas belonging to the cervical plexus are also well marked. I shall therefore leave the full consideration of the phenomena of anginal attacks for my next paper on the head and neck. For in order to understand their full significance we must first know the distribution of the segmental areas of the cervical plexus.

Thus to sum up the results yielded by a study of cardiac disease :—

(1) In diseases of the heart, and more especially in aortic disease, the pain is referred along the first, second, third and fourth dorsal areas.

(2) In angina pectoris the pain may be referred in addition along the fifth, sixth, seventh and even the eighth and ninth dorsal areas, and is always accompanied by pain over certain cervical areas.

Lungs.

For the production of visceral pain, and especially of tenderness in connection with the pain the disturbance in the organ has to be fairly acute. Now with regard to the lung we labour under several disadvantages if we attempt to find tenderness associated with its disturbances. For the majority of cases which occur in our hospitals are chronic,

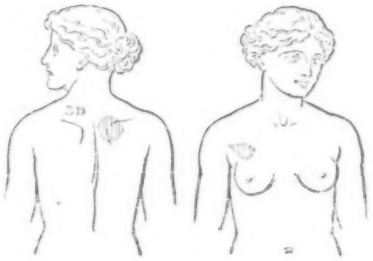


FIG. 23.

To show the areas of cutaneous tenderness in a case of acute bronchitis (Case No. 24).
These spots represent the maxima of the 2nd dorsal segment.

and those which are acute, such as lobar pneumonia, are not as a rule in a condition to give an account of themselves.

Fig. 23, however, shows the position of intense pain in a case of acute bronchitis. The pain was particularly bad on coughing. The areas in this figure also show the position of great cutaneous tenderness. The patient said she was so sore "she felt as if she had been beaten." The signs of bronchitis were far more marked on the right side than the left in this case.

Case 24.—Mrs. B., aged 34.

Pregnant seven months. Called to her on account of bad cough and pain in chest, which came on four days before. Temp. 103. Pulse 110. Respn. 30. Numerous mucous râles all over right side and also at left base. No dulness. Pain on coughing, associated with great cutaneous tenderness over the areas in fig. 23. Pressure over these areas, especially over the one in the back, seems to start the cough.

She subsequently aborted, and died June 8. No definite signs of pneumonia. Lochia normal. No pain or disturbance connected with uterus or other organs of the body.

With regard to phthisis which is the lung disease on which one has to depend, an enormous number of cases have neither pain nor tenderness. Some have pain and only a few have definite cutaneous tenderness. These as far as I have seen so far are those with an irritable cough in the early stage. Many will tell you they have suffered pain "but that has passed off now." Perhaps the commonest place is over the same areas as those in fig. 22. Another very common area to be affected is that of the sixth dorsal segment. The two main points of this area are situated the one posteriorly at the level of about the seventh dorsal spine just internal to the angle of the scapula and the other in front under the breast. The lowest area or the lowest two areas of the cervical plexus are also frequently affected. To these areas belong the tender points at the posterior aspect of the shoulder joint, and those over the second rib and under the clavicle. The inner side of the arm and the axilla are also sometimes affected, and not uncommonly the pain and even tenderness may extend down the ulnar border of the arm and forearm to the ulnar side of the hand.

The case mentioned on p. 34 shows the possible connec-

tion between the area of these referred pains and the area occupied by herpetic eruptions. A man of 34 years of age, with evident, though slight, signs of advancing phthisis at the right apex was accustomed to have pain and a sore feeling over the third dorsal spine and the spine of the scapula "whenever he caught cold" (*i.e.*, whenever his cough was worse). He came to me "having caught a fresh cold," and this time the pain to which he was well accustomed did not pass off but was followed by a herpetic eruption over the scapula and in the axilla corresponding to the area of the third dorsal segment.

Thus in diseases of the lungs the areas corresponding to the first, second, third, fourth, fifth, sixth and seventh dorsal segments may be affected. The second, third, fourth and fifth dorsal seem to be most commonly affected. There may also be pain and tenderness over the lower areas such as that of the seventh dorsal but in such cases there is frequently distinct evidence of dyspepsia, so common in the early stages of phthisis.

Œsophagus.

Stricture of the œsophagus affords one of the most definite instances of referred pain for here the pain can be produced by swallowing. In several cases the necessity for passing a bougie has been taken advantage of to discover the situation of the pain. As the bougie passes the patient is asked to mark out with a pencil the spots where the pain is situated. In most cases the pain is referred to a point about two inches from the middle line exactly at the angle of the scapula and is associated with a pain in front over the fifth space about half-inch internal to the nipple line. There is sometimes pain in the back over the sixth and seventh vertebral spines. Frequently pain thus referred is the only phenomenon but sometimes cutaneous tenderness is also present over the spots of referred pain.

One of the most marked cases I have seen was in a man of 65 years of age who in consequence of diet and treatment presented scarcely any pain. The stricture, which was fourteen

inches from the mouth, began to close and it was therefore determined to pass a bougie. After the passage of the instrument he complained of a good deal of pain and I found the whole of the fifth, sixth, seventh and eighth dorsal areas exquisitely tender. The curious upper border of the fifth dorsal area with its axillary flap was beautifully shown. By the next day this tenderness had subsided and was replaced by tender points only. These tender points in turn subsided one by one. He died a month afterwards of malignant disease of the œsophagus.

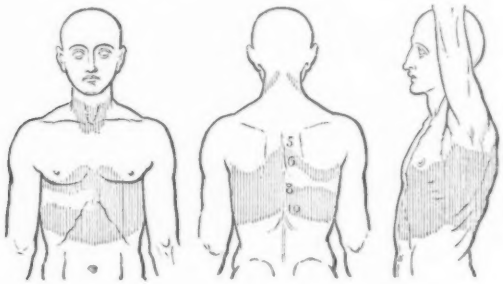


FIG. 24.

To show the areas of cutaneous tenderness in a case of stricture of the œsophagus, induced by the passage of a bougie (Case No. 25).

The areas represent the supply of the 5th, 6th, 7th, and 8th dorsal segments on the left side, and the 5th, 7th, and 8th dorsal on the right side. The upper border of the 5th dorsal is well shown on the lateral figure.

The numbers on the posterior view represent the spines of the respective dorsal vertebræ.

Case 25.—Samuel L., aged 64. (Mr. Heath, U.C.H.)

One month before admission difficulty in swallowing solids. Gradually getting worse. Now even liquids regurgitate. Healthy looking man. Somewhat wasted. When he attempts to swallow milk he makes efforts of deglutition for about 15 seconds and then milk comes up again.

Complains of pain at episternal notch and over a point in the fifth space just inside the nipple on both sides. Also sometimes in the back just internal to the angle of the scapula. Cutaneous tenderness more or less marked over all these points.

No physical signs of aneurism. Voice normal.

No physical signs in lungs or heart.

No enlarged glands. Liver not enlarged.

Took prepared foods well and became almost free from pain and cutaneous tenderness. June 8, an œsophageal bougie was passed

14 inches from teeth but it was found impossible to get it into the stomach. Tenderness the same evening as in fig. 24. Very similar results obtained on June 15 after similar abortive attempts to pass bougie.

Died in August of complete closure of maligant stricture.

As the bougie passes the patient frequently complains of "choking pains" at the episternal notch. I have occasionally, though rarely, found tenderness associated with this pain, marking out one of the lower cervical areas.

Thus the most prominent situation of pain and tenderness in stricture of the œsophagus corresponds to the distribution of the fifth dorsal, but the sixth, seventh and even the eighth dorsal segments are also frequently affected more or less.

The Breast.

Many factors combine to hinder the determination of the referred pain and tenderness due to disturbances of the breast. For it is notorious that the majority of tumours of the breast give no referred pain, and secondly, whenever a woman is run down, from whatever cause, she tends to suffer from a pain under the breast, associated with tenderness; moreover, the disturbance has to be fairly acute, or reference does not take place.

The most perfect example of referred pain in its purest form, was produced by retention of milk in one breast. Here there was marked cutaneous tenderness over the fourth and fifth dorsal roots. Fig. 25 was taken from a

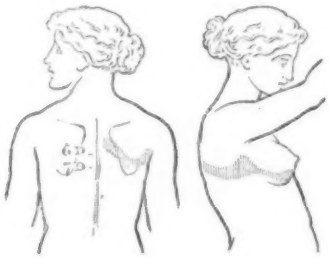


FIG. 25.

To show the distribution of cutaneous tenderness in Case No. 26. This area represents the distribution of the 5th dorsal segment.

young woman whose child was 4 months old. She suffered from sore nipples, and every time the child sucked she had pain over the angles of the scapulæ, over the posterior part of the axilla, and over the lower half of the breast. The tender area corresponded exactly with that of the fifth dorsal root.

Case 26.—Mrs. B., aged 34. Is suckling 4 months' old child. Right nipple was sore, and bled when the child sucked. This is now healed, but she complains of pain in the breast and pain in the back, between the posterior border of the scapula and the spine, on the right side, at about the level of the sixth or seventh dorsal spine. This pain is accentuated every time the child sucks. Marked cutaneous tenderness, as in fig. 25. No mastitis. Temperature normal.

It is not at all an uncommon thing for women who suckle children into the second year to complain of pain "over the tip of the shoulder blade." Mostly such women are debilitated and weakly, but I made the following observation in a strong healthy woman who was suckling an 18 months' child to prevent conception. She was free of pain or tenderness before she put the child to the breast, but after the child had pulled violently and vigorously at her breast for a few minutes, the pain over the shoulder blade began to appear. She let the child suck until he fell asleep, and by that time there was a distinct area of cutaneous tenderness over the lower border of the scapula. She was accustomed to let the child pull at the breast whenever he woke in the night, and she said that in the morning her shoulder blade was sometimes so sore she could not put on her stays.

Thus the principal pains and tenderness in disturbances of the breast gland and ducts (other than new growth) seems to be situated over the fourth and fifth dorsal roots.

Stomach.

Pain in gastric disturbances is almost always accompanied by some cutaneous tenderness. This tenderness lies in front between the level of the ensiform cartilage and the umbilicus. It occupies one or more of the three

areas corresponding to the supply of the seventh, eighth and ninth dorsal segments.

One of the commonest situations for pain is at the epigastrium, just over the ensiform cartilage, and a reference to plate IA, shows that this is one of the maximum spots of the sub-scapulo-ensiform, or seventh dorsal area. In cases of so-called gastric ulcer, cutaneous tenderness is nearly always present over this area and over a point in the line of the angle of the scapula at the level of the ninth dorsal spine.

The middle area of this group (eighth dorsal) also appears frequently, but as far as my experience goes, seldom alone in stomach affections. It is nearly always associated with the one above or the one below it. Its characteristic tender point is situated over the eighth space, almost exactly in the mid-axillary line.

The next area, corresponding to the ninth dorsal root, is one of the commonest of all the stomach areas. It appears repeatedly in cases of gastric ulcer, either alone or in combination with one or more areas of the group.

It is a curious thing that all these areas associated with the stomach tend to appear bilaterally. This was so in a case of which fig. 26 was taken. It represents the cutaneous tenderness which appeared in a youth who was suffering from a violent attack of stomach pain, accompanied by vomiting, as a consequence of irritant food.

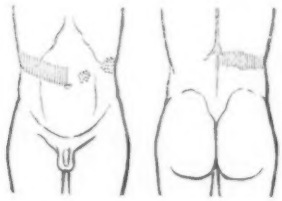


FIG. 26.

To show the distribution of cutaneous tenderness in a case of acute gastritis (Case No. 27). The area on the right side represents the full extent of the 9th dorsal area, whilst the spot over the costal border on the left side represents its most prominent maximum.

Case 27.—Youth, aged 14. Pain in the abdomen for the last twelve hours. Intense—keeps him doubled up. Points out position of the pain by placing the flat of his hand just above the umbilicus, with the fingers pointing to the linear alba. Cutaneous tenderness (blunt end of pin called "prick") over the area in fig. 26.

Bowels open. No constipation or diarrhœa. Has vomited several times.

Pain and tenderness gone next day.

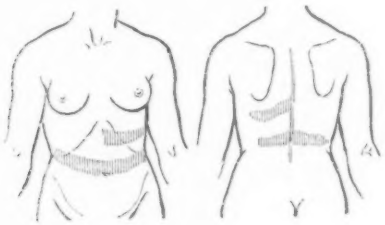


FIG. 27.

To show the distribution of cutaneous tenderness in a case of so called gastric ulcer (Case No. 28).

The areas on the left side represent the distribution of the 7th and 9th dorsal segments. That on the right side represents the distribution of the 9th dorsal segment.

Case 28.—M. E., aged 27, single, servant.

Two years ago attack of pain and vomiting. No blood. Recovered almost completely, but was subject to pain after food.

November 4, 1891, felt sick, and at 8.30 a.m., vomited large quantity of altered blood. Next day passed half pint of black stuff by bowel.

Constant cutaneous tenderness, mostly situated over the upper area; increased by food or by anything warm, but never quite absent. Pain referred to ensiform cartilage and to the back.

Vomits soon after food. Bowels confined.

Somewhat anæmic.

I cannot yet say whether the tendency for these areas to appear on the right side in stomach affections has any significance.

I do not think it is possible at present to say whether the appearances of one or other of these areas of tenderness has any value in differential diagnosis. However the uppermost one (seventh dorsal) seems particularly to appear in anything which causes vomiting [compare Case 1, page 11],

and when it appears as a sequel to vomiting it is frequently accompanied by the area next above it, corresponding to the sixth dorsal. Now we saw above that this sixth dorsal area was associated with disease in the lower part of the œsophagus, and hence I feel inclined to think that when both areas are present the disease is somewhere near the cardiac orifice. In the same way the ninth dorsal area is shared by both the stomach and intestine, and thus a lesion of the stomach causing pain over this area is probably situated nearer to the pyloric orifice; I possess, however, no records of *post-mortem* examinations to prove this, and at present it is a pure supposition.

In cases of gastralgia and gastic ulcer in which the pain bears a definite relation to the ingestion of food I think I am justified in making the following statement as regards the length of time after food at which it comes on. In those cases where the seventh dorsal segment is affected and the maximum tenderness is situated at and around the ensiform cartilage the pain comes on within half an hour after the food is swallowed. On the other hand in cases where the ninth dorsal segment only is affected the pain does not come on until the food has been swallowed at least an hour. This again tells in favour of the view that the seventh dorsal segment supplies the cardiac end of the stomach whilst the ninth dorsal supplies the pyloric portion.

Thus the areas which are affected in disturbances of the stomach correspond to the supply of the seventh, eighth and ninth dorsal segments.

Intestine.

The pain of intestinal affections is frequently quite diffuse and ill-defined. In fact it is not referred at all. Such pain is unaccompanied by tenderness in the skin. On the other hand a considerable number of cases of intestinal disturbance exhibit well marked pain and tenderness. I watched a large number of cases of diarrhœa which occurred in out-patient practice, and, as far as my experience goes at present, found that those cases in which "rolling," "griping," "doubling

up pain" was complained of there was no pain in the back and no cutaneous tenderness. On the other hand a fair number of cases of acute diarrhœa, either due to some direct irritant, such as improper food, or more rarely to the disease known as "summer diarrhœa" were associated with pain below the umbilicus and over the twelfth rib, marking out to a greater or less extent the area supplied by the tenth dorsal segment. In those cases associated with a direct history of irritant food, I found the area corresponding to the ninth segment was also tender. If vomiting occurred in association with this diarrhœa, all the areas from the seventh to the tenth dorsal inclusive, might be present to a greater or less extent.

On the other hand the majority of cases of summer diarrhœa in which the stools were like "black water" complained of no referred pain, and did not show cutaneous tenderness.

It is also noticeable that cases of lead colic do not complain of referred pain and do not show cutaneous tenderness, even when the onset is fairly acute, and the "griping" pain well marked.

I feel inclined to think the pain and aching in the back loins and abdomen which appear in typhoid fever, is sometimes referred intestinal pain. In the case 52, there was at one time well-marked tenderness in the skin corresponding to the 10th dorsal area only. I have since seen several cases of typhoid fever, in which there was well-marked cutaneous tenderness over the abdomen and loins on both sides at the end of the second week of the disease.

Fig. 28 is taken from a man of thirty-nine, who suffered from dysenteric diarrhœa with fluid dark brown offensive stools, frequently containing blood. He was much relieved by enemata of creolin, but just after the injection he suffered a good deal of pain accompanied by exquisite cutaneous tenderness as shown in the figure. So tender was his abdomen, that he was obliged to have the clothes supported by a cradle. The tenderness varied from time to time; sometimes it occupied only the area of the tenth dorsal segment, sometimes that of both the tenth and

eleventh segments. Pressure relieved this pain, and to show how purely superficial it was, I may mention that it was completely removed by an ointment containing thirty grains of cocaine to the ounce of lanoline.

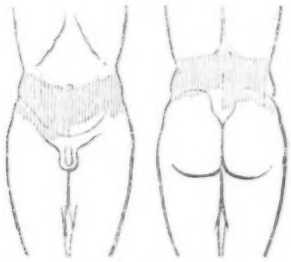


FIG. 28.

To show the distribution of cutaneous tenderness in Case No. 29. The area on the left side represents the 10th dorsal; that on the right side the 10th and 11th dorsal.

Case 29. Edward B., aged 39, compositor. (U. C. H., Dr. Roberts).

Eight months before admission had an attack of diarrhœa in which he passed blood. Lasted six weeks. Diarrhœa never quite ceased.

On admission pale, very thin, skin dry.

Appetite good, tongue large, flabby, not furred. No nausea or vomiting. Stools fluid, dark brown, very offensive. Contained altered blood in considerable quantities. Sometimes stools were quite fluid, sometimes they contained a few hard lumps. Abdomen distended.

No physical signs in lungs or heart.

Complained of a great deal of pain in the abdomen below the umbilicus, and in the back below the twelfth dorsal spine.

There is great cutaneous tenderness, worse at one time than at another. Always situated in the areas of the tenth and eleventh spinal segments, but sometimes worse on one side, sometimes on the other. So tender is he, that the bedclothes are supported by a cradle. Yet firm deep pressure relieves, rather than aggravates, his pain.

Under creolin enemata and oil injections he improved, and left the hospital, passing liquid stools free of blood and free from pain. Tenderness absent.

Died at home two months afterwards.

In this connection a case of malignant stricture of the splenic flexure (autopsy) is instructive. The maximum tenderness was over the tenth dorsal with some tenderness over the eleventh dorsal area. These areas were I take it, of intestinal origin. In addition she suffered from very marked dyspepsia with flatus and occasional vomiting, and both the seventh, eighth and ninth areas are more or less tender (stomach). In addition she developed bronchitis with troublesome cough and expectoration, and it was from this that she actually died. With the advent of bronchitis she developed great tenderness in the upper part of the back, between the spine of the scapula and the third to fourth vertebral spines, the point corresponding to the second dorsal area.

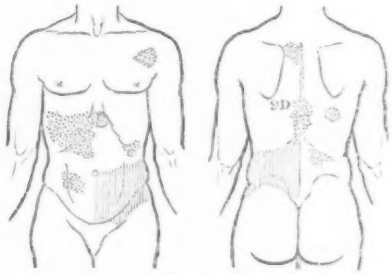


FIG. 29.

To show the distribution of cutaneous tenderness in a case of stricture of the sigmoid flexure of the large intestine (Case No. 30). The shaded area represents the 10th and 11th dorsal. The dotted areas are the same on the opposite side, and represent its maximum spots. The dotted area in the region of the liver represents the 7th and 8th dorsal. The dotted areas in the region of the spine of the scapula, and the front of the chest represent the maxima of the 2nd dorsal.

Case 30.—E. W., aged 46, U. C. H. (Mr. Heath), June, 1891.

Four years ago began to feel "fulness" in lower part of bowel.

Three years ago motions began to get smaller and passage gave her pain. Constipation alternating with diarrhœa.

November, 1890, passed blood from rectum. This has continued on and off ever since.

Losing flesh lately.

On admission abdomen much distended. Marked cutaneous tenderness over left half of abdomen below umbilicus as in fig.

28. This was probably due to the distension of the gut produced by the stricture and not to the destruction caused by the malignant disease itself. Some ill-defined tenderness on right side. Liver felt 3in. below costal border.

Considerable cutaneous tenderness in the region of the liver and back as in fig. 29.

Early pneumonic signs. Large râles and increased vocal resonance, especially left side. Much cough and some expectoration.

Note the spots of tenderness in the upper part of chest.

June 17. Difficulty in passing urine, and diarrhœa came on.

August 1. Died somewhat suddenly.

Post-mortem.—Intestines matted with recent lymph. Fluid in abdomen. A large mass of new growth involves sigmoid flexure. Large intestine and cæcum distended. Liver much enlarged; many small secondary growths. Gall bladder contains large number of stones. Left pleura full of fluid. Left lung collapsed.

Thus a certain number of cases in which the intestine from the pylorus to the end of the colon was affected showed definite cutaneous tenderness, corresponding more or less to the ninth, tenth, eleventh and possibly twelfth dorsal areas.

Liver and Gall Bladder.

The pain of liver disease is difficult to work out satisfactorily, for in those cases which are accompanied by pain the diagnosis of uncomplicated liver disease is not always satisfactory. Moreover a large number of cases of disease of the gall bladder which come to operation or autopsy are in reality secondary to malignant disease. In this connection the following case, for which I am indebted to Dr. Ringer, is very instructive. He was aged 47, and in July '91 began to suffer for the first time from severe attacks of biliary colic followed by jaundice. In January he was admitted, suffering from considerable pain, with cutaneous tenderness over the eighth and ninth dorsal areas on the right side, and the sixth and eighth dorsal areas on the left side. The tenderness was well defined, and on his left side the curious relation borne to one another by the sixth and eighth dorsal areas in the axillary line was beautifully shown. He was not jaundiced, but there was a trace of bile

in the urine. A mass could be felt under the right costal margin, which was rightly supposed to be gall bladder. Mr. Pollard operated and removed 138 gall stones. All the old pain and tenderness disappeared and never returned, but secondary growths made their appearance in the lungs and skin, and he died on February 11th. A large mass of new growth was found in the portal fissure, blocking the bile ducts. Now although there was malignant disease I cannot help thinking that the pain and tenderness were due to the gall stones, for the pain exactly resembled that in cases of biliary colic in which gall stones pass, and it disappeared entirely after the operation although the malignant disease was untouched.

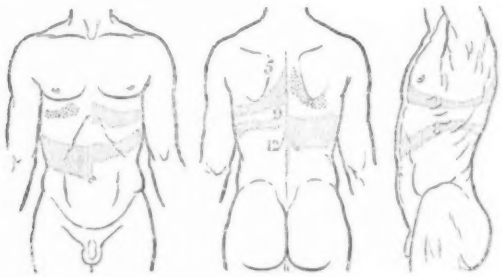


FIG. 30.

To show the extent of cutaneous tenderness in a case of hepatic colic (Case No. 31).

The areas represent the distribution of the 6th and 8th dorsal on the left side, and the 8th, 9th, and part of the 6th dorsal on the right side.

The figures on the posterior view are placed at the level of the spines of the vertebræ. The figure 8 on the lateral view is placed over the 8th intercostal space.

Case 31.—Frederick C., aged 47, tailor. U. C. H. (Dr. Ringer). *July*, 1891. Several attacks of gallstone colic followed by jaundice. The attacks came on daily or there might be a week between the attacks. In the intervals pain in the right side. Stools greyish white during the attacks. Was perfectly well up to July.

Jan. 1892, admitted. Emaciated and pale. No jaundice. Continuous aching pain in right hypochondriac region. Tongue furred. Liver not felt. Below ninth costal cartilage an indistinct mass can be felt. Bowels confined. Motions normal colour. Urine contains a trace of bile. No vomiting.

Intense cutaneous tenderness so great that the head of a pin is felt as a sharp point, and he says that he is being pricked when touched over the areas marked in fig. 30.

Jan. 9. Mr. Pollard operated. Extensive adhesion round gall bladder.

Jan. 14. Mr. Pollard removed 138 gall stones from the bladder.

Jan. 15. All old pain gone. No sign of cutaneous tenderness. Slight pain in the wound only.

Feb. 9. Cough. Pneumonic signs left lung.

Feb. 11. Died.

Post-mortem.—Bile ducts surrounded by dense firm tissue. At portal fissure large mass of new growth. Growth limited very closely to Glisson's capsule. No scattered masses in liver. No new growth in gall bladder. Common bile duct and cystic duct were patent. Numerous small growth in omentum. Stomach, intestine, spleen, kidneys normal. Lower lobe of left lung solid. Pneumonic patches; gangrene of extreme lower part.

The well known pain in the back at the level of the eleventh dorsal spine extending outwards to the line of the angle of the scapula and associated with pain "over the gall bladder" are simply the maximum spots of the eighth dorsal area. The eight dorsal segment seems to be particularly associated with the gall bladder and I believe it is for this reason that this area shows a greater readiness to appear on both sides in biliary colic than any of the other areas associated with the liver.

In this description of the pain and tenderness in association with gall stones I am in almost complete accord with Mackenzie.

In some cases of liver disease such as the enlarged and congested liver of primary, and not of secondary, origin the tenderness takes one segment lower than in cases of gall stones. The whole of the tenth dorsal area is more or less tender. Makenzie expresses a doubt as to whether this is not due to perihepatitis but I think I have satisfied myself that there is this cutaneous tenderness between the twelfth rib and the crest of the ilium. These cases also show the spot in the eighth space and mid-axillary line so common in liver disease.

Again, disturbances of the liver are associated with pain and sometimes tenderness over the shoulder joint and over

the neck corresponding to the distribution of the lower roots of the cervical plexus; but I have not yet satisfied myself of the diagnostic significance of this pain in the lower part of the neck.

Thus in disturbances of the liver the pain is referred to, and the tenderness appears over, the areas supplied by the eighth and ninth dorsal segments and sometimes over that of the tenth dorsal on the right side. If the gall bladder is affected the pain and tenderness also lie over the eighth and ninth dorsal areas but the fifth, sixth and seventh are as a rule also implicated more or less.

Kidney and Ureter.

The pain of renal calculus is most useful for this investigation; for the diagnosis of stone in the kidney is practically always confirmed by operation.

The pain is said to be situated about the tip of the twelfth rib in the loin behind, and over a point in the abdomen internal to the crest of the ileum. During an attack of colic the pain is said to shoot round "the lower part of the back" (about the first lumbar spine) to the front of the abdomen and thence to the thighs and even down to the inner side of the knee. This pain is associated with marked cutaneous tenderness especially after an attack of colic. After an attack the patient complains that he feels "bruised, just as if he had been beaten about the back and loins." Mostly the area of tenderness is the tenth dorsal but sometimes the eleventh and twelfth dorsal areas are affected. Occasionally even some portion of the first lumbar with its spot on the inner side of the knee is marked out in tenderness.

One patient gave me the following interesting account showing the difference between an attack of renal colic in which no stone passed and one in which he actually passed a stone.

I first saw him just after an attack. The pain was located over the tip of the twelfth rib and on part of the crest of the ileum. The whole loin was sore and tender over an area marking out the tenth, eleventh and twelfth dorsal areas. No calculus had passed. Sometime before this he

had had an attack which was similar excepting that the pain gradually spread from the region of the tenth dorsal to the upper sacral region and to a point in front just above Poupart's ligament 2 to 3 inches from the middle line (maximum of eleventh dorsal). Then it spread down the inner side of the thigh to the knee; then to "the bottom of the back" through the perineum to the urethra. In this attack his doctor said he passed a uric acid calculus.

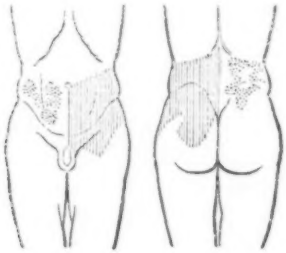


FIG. 31.

To show the distribution of cutaneous tenderness in a case of severe renal colic (Case No. 32).

The shaded area on the left side represents the 10th, 11th, and 12th dorsal. The dotted area on the right side represents the less defined tenderness associated with the presence of the maxima of the 10th and 11th dorsal areas.

It is interesting to note that this case showed a rather wider distribution downwards [11th and 12th dorsal] than is usual in cases of renal calculus in the inter-paroxysmal period. This was possibly due to the impaction of the stone in the ureter.

Case 32.—Thos. D., aged 37. U. C. H., June 30, 1891 (Mr. Hill).

Eight years ago passed a stone of a slate colour and the size of a bean. Pain came on after a long railway journey and lasted for twenty-four hours when the stone passed.

Was free from pain for a year and then began to pass crystalline masses, some of which were the size of a pea and were covered with minute points. On analysis they were said to be uric acid.

For three years he had about one attack of pain a year and passed small calculi. Lately the attacks have been more frequent. Blood always appears in urine after passage of calculus.

When I saw him he had just had an attack of colic, but no stone had passed. The pain starts behind at about the tip of the twelfth rib and runs forwards to a point in the left iliac fossa. Thence it passes to an intense centre of pain situated just above the internal ring. Thence it passes to another very painful focus

in the pubic hair close to the root of the penis. Thence it runs down the inner side of the thigh to end at about the lower third of the inner aspect above the knee. As soon as the pain reaches the point over the pubes retraction of the testicle takes place. He says the pain is like that of cramp and at the same time there is pain as if he had taken a strong purgative.

During the paroxysm certain points are excessively sore. The testicle is very painful and tender, but he says the scrotum is never sore.

When I first saw him he was suffering from great pain in the left loin and lower part of abdomen and groin.

There was marked cutaneous tenderness with exaggerated superficial reflexes on the left side as in fig. 31. There was also some tenderness of a less defined character in the right loin and abdomen.

This cutaneous tenderness does not affect the scrotum except perhaps at its extreme upper part, *i.e.* the greater part of the skin of the scrotum is not tender. But the left testicle is intensely tender, and he cannot bear even the slightest touch.

July 1. Mr. Hill removed a calculus weighing six grains from the left ureter.

Seen again April, 1892. Wound healed. Has remained free from pain. No tenderness.

Case 33.—Geo. H., aged 24. U. C. H., June, 1891 (Mr. Hill).

Pain in left loin and across front of abdomen for five to six years. Agonizing attacks about once a month. Blood in urine, especially after an attack.

On admission.—Abdomen flaccid. Lower end of left kidney felt; does not seem to be enlarged.

Urine: amber coloured; acid. Microscopically contains blood and urate of soda.

The pain radiates from the tip of twelfth rib behind to a point in the left iliac fossa about midway between the anterior superior spine and the umbilicus and a little below the level of the umbilical line.

Definite cutaneous tenderness over the area of the tenth dorsal spinal segment. The tenderness is especially marked over the tip of the twelfth rib behind, and over the point on the left iliac fossa to which he refers his pain.

June 17. Mr. Hill removed an oxalate of lime calculus from the left kidney. It lays free in the pelvis and weighed twenty-seven grains. Dark coloured, ovoid stone, flattened from side to side and studded with small nodules.

Oct. 28. Has remained free from pain. No tenderness. Is in excellent health. Urine normal. No blood. Scar looks perfect.

In a patient with a floating kidney, who had no pain or tenderness as he lay in bed, I have seen the characteristic pain at the tip of the twelfth rib with cutaneous tenderness

over the area of the tenth dorsal, produced by the rough manipulation of an examination.

Thus the kidney is particularly associated with the cutaneous areas of the tenth, and to a less extent those of the eleventh and twelfth dorsal and first lumbar areas. On the other hand, diseases of the pelvis of the kidney and of the ureter, seem to be particularly associated with the eleventh and twelfth dorsal and first lumbar areas.

Testis.

The testis is not so satisfactory as the kidney, owing to the great tendency of diseases of this organ to produce "spreading." (*Vide* chapter iv.)

In the case of a youth in whom one testicle had not descended, but lay in the inguinal canal, I made the following observations. In the course of his work he knocked it against a bench and it swelled and became painful. The pain was situated at the tip of the twelfth rib behind, and just internal to the crest in front, oddly enough on the opposite side, mainly, to the affected testicle. There was great superficial tenderness over the area of the tenth dorsal segment.

Case 34.—George D., aged 16. U. C. H., Jan., 1891 (Mr. Barker).

Is a carpenter, and pressure on the bench causes great pain in the undescended right testicle.

No testicle in right half of scrotum, but just to the inner side of the external iliac artery is a soft roundish swelling. When pressed upon it gives him testicular sensation. When he stands up index finger can be inserted into external abdominal ring, and the lower end of the swelling can be distinctly felt. In the erect posture it comes lower and can be pressed against the crest of the pubes. This gives him great pain.

The testicle itself is very tender, but the cutaneous tenderness on the occasion on which I saw him marked out the tenth dorsal area on the left side (not the right as might have been expected).

Mr. Barker removed the testicle, and he left the hospital Feb. 5, free from pain.

Again in cases of true traumatic orchitis when acute, there is intense pain over the loin and sometimes tenderness in the area of the tenth dorsal segment.

Thus the testicle seems to be peculiarly associated with the tenth dorsal spinal segment.

Epididymis.

Epididymitis is mostly gonorrhœal in origin and is associated with considerable pain when acute. In an ordinary moderately acute case the pain is situated over the fifth lumbar and first sacral spines behind and just over about the internal inguinal ring in front. These spots are generally tender and correspond to the maximum of the eleventh dorsal area. Sometimes the pain and tenderness are as in fig. 32, and thus correspond to both the eleventh and twelfth areas.

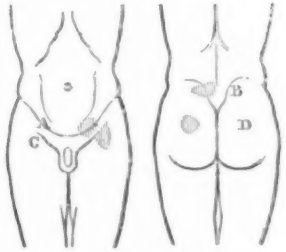


FIG. 32.

To show the cutaneous tenderness, and at the same time the seat of the pain and aching in a case of gonorrhœal orchitis (Case No. 35).

The spots marked A and B, correspond to the maxima of the 11th dorsal area. The spots marked C and D correspond to the maxima of the 12th dorsal area.

Case 35.—S. K., aged 28. U. C. H., casualty department, May, 1892. Had a "running" for nine weeks. It stopped a week ago, and next day swelling came on in "privates." Now well marked epididymitis. Left epididymis swollen to size of small apple, and very tender.

Pain passes from the point D (fig. 32) to B, and thence round the crest to the lower part of the abdomen and upper part of the groin. It is not sharp or shooting, but is dull and aching, "as if someone had kicked him." Cutaneous tenderness, as in fig. 32.

It is a noticeable and instructive fact that although the epididymis is exceedingly tender to manipulation, there is no cutaneous tenderness of the scrotum. Thus there is fre-

quently considerable cutaneous tenderness over the outer border of Scarpa's triangle in a situation far removed from any of the affected structures, but no cutaneous tenderness over the epididymis itself [*i.e.*, scrotum], excepting at the upper portion, where the skin is supplied from the twelfth dorsal segment.

If the epididymitis is very acute, the tenderness may become very extensive, reaching to the inner side of the knee and the outer side of the thigh, and it may even spread into the perineal area, jumping from the lumbar supply to that of the sacral areas.

Case 36.—Peter W., aged 40. U. C. H. (Mr. Heath), August 30, 1891. Denies gonorrhœa or syphilis.

August 27. Gnawing pain in left testicle.

August 30. Considerable swelling of left testicle, both body and epididymis. Epididymis hard, nodular, tender. Body also swollen, a little harder than natural and very tender. Spermatic cord as a whole thickened. Skin of scrotum reddened, and a little œdematous. No urethral discharge. Prostate normal in size and consistency. Temperature rose to 100.6.

Pain starts from the lower lumbar and upper sacral region, extending laterally on to the buttock. It also extends down the outer and inner aspects of the left thigh, and on to the abdomen above the pubes.

Intense cutaneous tenderness marking out the eleventh, twelfth and first lumbar areas.

September 7. All pain gone excepting in groin. No marked tenderness.

Thus the epididymis seems to be particularly associated with the 11th and 12th dorsal segments.

Bladder.

Erichsen states that the pain of stone in the bladder "may be experienced not only in the region of the bladder and perineum, but may radiate widely in the course of the nerves of the lumbar and sacral plexus, the patient complaining of a heavy dragging sensation in the groins, extending down the outside and back of his thighs and not uncommonly experienced in the soles of the feet."

I have met with no case, so far, which showed this wide distribution. The patient usually complains of pain in the

perineum—pain at the end of the penis and pain over the ischial tuberosities. Sometimes this pain is accompanied by tenderness. Fig. 33 shows that this pain represents the gluteo-perineal (third sacral) area, for the patient complains exactly of this pain, and the tenderness occupied the area in the figure. The pain was caused by the unfortunate removal of a piece of mucous membrane of the bladder, during the crushing of a phosphatic calculus.

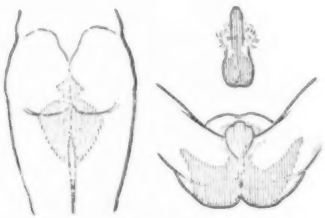


FIG. 33.

To show the cutaneous tenderness in a bladder case (Case No. 37).
The shaded areas represent the 3rd and 4th sacral. Note that there is a small patch of skin at the base of the scrotum which is unaffected. This is supplied from the 12th dorsal.
Compare with this figure the distribution of the eruption shown in fig. 10.

Case 37.—Wm. Thos. C., aged 51. U. C. H., May, 1892. Twenty years ago gonorrhœa. Two years afterwards developed a stricture. Up till six months ago had been in the habit of passing a catheter.

April 24. Could not pass any urine. No instrument could be passed into the bladder. Under opium and hot baths, No. 5 French passed the same evening. Urine alkaline, foul mucus, pus crystals of triple phosphates and micrococci.

He was slowly dilated, and on May 23 calculi were detected in the bladder.

May 24. Complains of pain before and during the passage of urine, but none afterwards. The pain is situated a little to the left of the anus and in the perineum. There is no cutaneous tenderness.

May 25. Two small stones ($\frac{3}{4}$ inch and $\frac{1}{2}$ inch in diameter) were crushed. The second stone gave considerable trouble, and on more than one occasion a small piece of mucous membrane was brought away in the blades of the lithotrite. Bleeding was considerable.

May 26. 9 a.m. Rigor.

May 27. Frequency of micturition; every hour and a-half to two hours. No blood; some pus and mucus.

May 28. Frequency of micturition continues. He complains of over the following points:—

[1st] Over the coccyx.

[2nd] Over a point on the inner aspect of both thighs, just external to the outer border of the perineum.

[3rd] Pain also in middle of perineum, "running down the passage" to the tip of the penis.

There is no pain above the pubes.

There is intense cutaneous tenderness in the extreme lower part of the spine, and in the perineum, as in fig. 33. The skin of the penis and scrotum is tender. Temperature normal.

May 29. The cutaneous tenderness was marked out again, and coincided with that in fig. 33.

June 3. Temperature normal since May 27. Another small stone (size of hazel nut) was crushed. Several small portions of soft tissue were removed from the bladder by the evacuator, evidently pieces of mucous membrane which had remained in the bladder since last operation.

Tenderness over a similar area was also produced by the accidental use of carbolic lotion instead of boracic, to wash out a patient's bladder. The whole glans, which had not been touched by the fluid, was intensely sore. Thus the ordinary pain due to lesions of the mucous membrane, seemed to correspond to the areas of the fourth and third sacral segments.

In cases of retention, however, an additional pain is found. Suppose a patient suffers from simple obstructive retention, the result of a stricture in a man, or better still, the tilting forwards of a fibroid or of the pregnant uterus in a woman, the bladder gradually fills, and a dull aching pain appears over the lower part of the sacrum, passing into the perineum. This pain is more or less continuous, and is described by women who have borne children as like the pain "at the very bottom of the back" in labour. Every now and then the patient is overcome by an irresistible desire to pass water and strains against the obstruction. And now a quite different pain appears, of a sharp, shooting character, radiating from the lower lumbar and sacral regions behind, round the ileum to the lower part of the abdomen just above the pubes, and also down the inner side of the thigh to the knee. This lumbar pain soon goes, only to

return again after an interval; but the sacral pain seems to be continuous. In all my cases the pain was completely relieved when the urine was drawn off. One of my female patients had retention several times, owing to the displacement of a large loose fibroid, causing pressure on the neck of the bladder. Each time she gave exactly the same description of the dual nature of her pain. She was always relieved by the passage of a catheter. The amount drawn off varied on different occasions from 40 to 60 fluid ounces.

Case 38.—Mary D., aged 34 years, married. U. C. H., obstetrical department.

August 22. Complete retention of urine for nearly twenty-four hours. Bladder reached to within an inch of the umbilicus.

She was in intense pain. The pain radiated from the lower part of the sacrum and coccyx into the perineum. It resembled a labour pain, and was accompanied by a feeling of weight and "bearing down" in the perineum.

When she strained to pass water this pain was joined by a pain over an area extending from the region of the fifth lumbar and first sacral vertebræ outwards, between the great trochanter and the crest of the ileum to the lower part of the abdomen and groin. It also extended on to the outer side of the thigh, and also down the inner side of the thigh to a point a little below the junction of the middle and lower thirds of the femur. This pain rapidly passed off, but recurred again and again whenever she tried to pass water.

No cutaneous tenderness.

Forty-one ounces of urine drawn off. Pain immediately relieved.

A large fibroid was felt, which had fallen forwards over the neck of the bladder.

She came up on two subsequent occasions, with retention from the same cause, and always complained of the two forms of pain.

Thus lesions of the mucous membrane of the bladder affect the lower sacral areas (third and fourth), whilst the pain of ineffectual contraction is difused over the eleventh and twelfth dorsal and the first lumbar areas.

Prostate.

Before I leave the male urino-genital tract I must mention the pain caused by diseases of the prostate. I have reserved this section to the last owing to the wide distribution of the pain in diseases of this organ.

The most marked pain is, as usual, caused by acute inflammation of the prostate—acute prostatitis.

The highest pain is situated over the twelfth rib behind and just internal to the crest in front. Pain may be present over the fifth lumbar spine behind, and also over the internal ring area in front, but I have no case where any lower dorso-lumbar areas were affected.

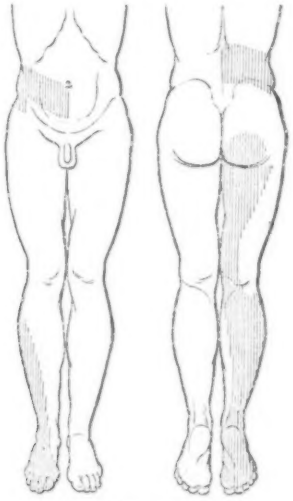


FIG. 34.

To show the distribution of cutaneous tenderness in a case of acute inflammation of the right lobe of the prostate (Case No. 39).

The uppermost area represents the supply of the 10th dorsal segment. The area on the back of the leg represents the 3rd, 2nd and 1st sacral from above downwards, and the area on the outer side of the leg the 5th lumbar.

But the patient also has pain over the lower part of the sacrum, and the ischial tuberosities over the upper part of the calves and over the soles of the feet, especially at the root of the great toe. One of my patients whose prostatitis was set up by improper instrumentation at the hands of a quack, said, "My life is a burden for I cannot stand owing to the pain in the soles of my feet, I cannot walk owing to the pain in the calves, sit on account of the pain over the

ischial tuberosities and in the perineum, or even lie owing to the pain in my loins and side."

If the prostatitis is fairly acute tenderness is present over all the areas corresponding to this distribution of pain—the tenth and eleventh dorsal, the first, second and third sacral.

In the case from which fig. 34 was taken, the tenderness was very well marked and the surgical report was "right side of prostate only, enlarged and tender."

Case 39.—Thos. T., aged 52, shampooer, casualty U.C.H., August 23, 1891.

Five weeks before he had retention of urine from a large prostate. No. 10 catheter passed. Came up August 21st, second attack of retention. Catheter again passed. Next day great pain. August 23, pain referred to spots in fig. 34, with cutaneous tenderness. Right lobe of prostate swollen, enlarged, and very tender. No prostatic discharge. Six weeks afterwards under treatment had lost all pain and tenderness in the skin and right side of prostate had subsided.

Thus the pain and tenderness when present in prostatic disease seem to belong partly to the dorsal and partly to the sacral areas. The dorsal areas most often affected are those corresponding to the tenth and eleventh dorsal segments. The lumbo-sacral areas which are affected correspond to the first, second and third sacral with sometimes the fifth lumbar in addition.

Ovary.

Ovaritis is the bugbear of the gynecologist owing to the general disturbances it is liable to produce. I shall therefore not describe the pains of ovaritis in detail, but only such pain as can be distinctly traced to the ovary.

During vaginal examination no pain is caused by taking a normal ovary between the fingers but a curious sensation is produced apparently akin to "testicular sensation." If however the ovary is inflamed very definite pain is produced which the patient localises in the loin behind and pain is also frequently referred to a point just below and to one side of the umbilicus. This pain is frequently accompanied by tenderness of the skin over the area of the tenth segment. When obstetric assistant I frequently found cases of ovar-

itis quite without tenderness. But after examination these same cases often showed marked cutaneous tenderness corresponding to the tenth dorsal area and to that area only.

Case 40.—Annie D., married. U.C.H. (Dr. Spencer) Aug. 1891.

Well developed healthy woman.

Nothing felt from abdomen. Body of uterus retroverted. Both ovaries prolapsed and easily felt. Both tender, right one more so than left.

Pressure on right ovary causes pain in the loin a little to the right of the middle line at the level of the second lumbar vertebra and pain in front about 1½ inch to the right and 1 inch below the umbilicus.

Cutaneous tenderness especially after examination accurately marking out the supply of the tenth dorsal segment. It shows the little downward tag just external to the anterior superior spine on both sides. The lower border of the tender area is 2 inches above the pubes in the middle line.

Case 41.—Mrs. V. U.C.H. (Dr. Spencer), Aug. 21, 1891.

Constant pain in left loin and in abdomen below the umbilicus. At menstrual periods has pain in addition over the lower part of the abdomen and down the back of the leg.

A prolapsed ovary is felt lying directly behind the uterus. It is excessively tender. Even the lightest pressure causing pain. This pain is referred to a point in the left loin corresponding roughly with the tip of the twelfth rib. It is also referred to a spot in front a little to the left of and below the umbilicus.

Intense cutaneous tenderness marking out in a beautiful manner the tenth dorsal area.

Thus the ovary like the testis causes referred pain and cutaneous tenderness along the tenth dorsal area.

The Appendages.

It is needless to mention that pressure on normal tubes causes no pain. But in some cases of salpingitis very definite pain is caused by manipulation. This pain is situated over about the fifth lumbar and first sacral spines and "in the groin" above Poupart's ligament.

After removal of the ovaries the stump is sometimes tender. In the case I have figured fig. 35, some ovary and the whole tube was left on the one side (right) and the whole ovary and part of the tube removed on the other side.

Spontaneous pain was almost completely absent and the patient was bright and intelligent. After examination she had some pain and developed the tenderness over the areas in the figure (fig. 35). Thus pain and tenderness from the ovary seem to follow a line corresponding to one segmental area higher than that from the tubes. This I believe to be a valuable point in diagnosis.

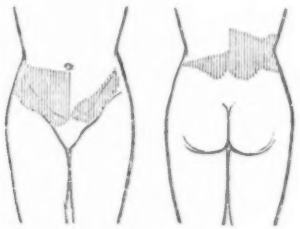


FIG. 35.

To show the cutaneous tenderness in Case No. 42.
The shaded area on the left side represents the 11th dorsal; that on the right side the 10th and 11th dorsal.

Case 42.—Eliz. B., widow. U.C.H. (Dr. Spencer), July, 1891.

In January, 1891, an attempt was made to remove both ovaries and both tubes for tubo-ovarian hæmatocele. The left ovary and greater part of the left tube were removed whereas a portion of the right ovary was left behind.

The uterus is movable, retroverted, and tilted a little to the right side. Right stump can be felt as big as tip of forefinger. On making pressure high up on the right side a tender body can be touched. Cervix rather small.

Strong, healthy looking woman, well nourished, bright, cheerful and not emotional. She complains of occasional pain, worse on right side than left in lower part of abdomen and back. Cutaneous tenderness well marked as in fig. 2.

Subsequently the whole of this pain and tenderness disappeared and she is now bright and well.

It is well known that after the removal of an ovarian tumour the patient complains of pain in the back over the region of the loin for which morphia is frequently given. It is customary to place a small pillow under the loin to ease this pain. In a case in which a tubal fœtation was removed by operation the tube and broad ligament were much

injured and the nurse called my attention to the fact that the pain was not in the usual place but lower down than usual over the upper part of the sacrum. I made the same observation in a case where a cyst had been removed from the broad ligament. Both these cases had borne children and described the pain as like a "dull labour pain." I subsequently asked several patients from whom true ovarian tumours had been removed whether their pain resembled labour pain and got a distinctly negative answer.

Thus the Fallopian tubes, like the epididymis seem to be particularly associated with the 11th and 12th dorsal segments.

Cervix and Os Uteri.

It is a well recognised fact that erosions and early malignant disease of the vaginal surface of the cervix produce no pain. But directly they set up cervical catarrh or begin

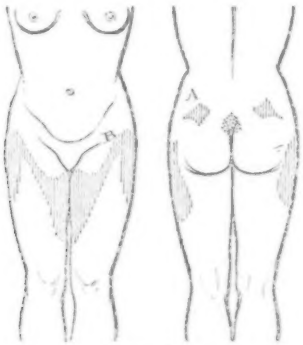


FIG. 36

To show the cutaneous tenderness produced by the passage of a probe in a case of cervical catarrh (Case No. 43).

The shaded areas marked A and B represent the 1st lumbar. The small dotted area over the sacrum represents the maximum of the 4th sacral.

to invade the broad ligaments pain begins. Such a disease was therefore too wide reaching in its effects for my purpose.

I made several interesting observations during the copper sulphate treatment, in cases of cervical catarrh. The effects

produced by the passage of the Playfair's probe were very instructive. As the probe enters the external os the patient complains of no discomfort. As it ascends the canal she begins to complain of a pain over the lower sacral region. As the probe advances still further the pain alters in position and is situated over the last lumbar and first sacral spines and in the groin in front. One of my patients repeatedly said:—"I first have pain at the very bottom of my back shooting through my legs and then as you push it further in I get a pain in my groin." The treatment was not in any sense of the word truly painful but it was frequently followed by some cutaneous tenderness in cases of cervical catarrh. (*Vide* fig. 36.)

Case 43.—Ellen T., aged 21, one child. U. C. H. (Dr. Williams), October, 1891.

Bright, well nourished, healthy looking woman.

Uterus, normal size. Slightly retroverted and retroflexed, but freely moveable. Right ovary can just be touched; not tender. Erosion on cervix. A good deal of clear mucus from cervical canal.

The erosion and cervical catarrh rapidly improved under the copper-sulphate treatment, and rest in bed.

October 22. Dressing the cervix was followed by cutaneous tenderness over the areas in fig. 36. She is now nearly well.

November 26. Free from pain and tenderness. Erosion healed. No discharge from cervix. Has passed through a menstrual period without pain. Has been at work for a week, and is bright and well.

In those cases where the canal is widely lacerated by previous labours, the lower (sacral) pain seems to be absent. In a case where a mucous polypus was very high up, not extending through the external os, the pain, accompanied by marked cutaneous tenderness, was situated over the area on the skin corresponding to the eleventh dorsal area together with the maxima of the twelfth dorsal.

This opens up the whole interesting question of dysmenorrhœa and its treatment, which I shall touch upon shortly later in this paper. (*Vide* p. 106.)

Thus the lower portion of the cervical canal seems to be associated with the 3rd and 4th sacral areas, whilst the upper portion in the region of the internal os is related to the areas of the 11th and 12th dorsal segments.

Pains during and after Labour.

During the first two stages of labour, pain appears over two separate points. The so-called "niggling pains" cause pain over the upper part of the sacrum and in the groins, whilst a pain appears later over the extreme lower part of the back in the region of the sacrum and coccyx.

The following case in which I induced labour at seven and a half months shows this very well, and gives us the clue to the meaning of these pains. The thin bougie was introduced in the evening, and I left her free from pain.

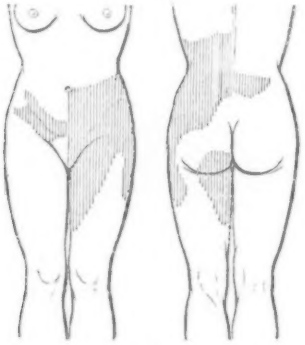


FIG. 37.

On the right half of the body on each of these figures is shown the cutaneous tenderness which appeared during the second stage of labour in Case No. 44. The area marked out represents the 11th dorsal.

On the left half of the body in each figure is shown the cutaneous tenderness which accompanied the "after pains" 48 hours after delivery in the same case. The areas represent the 10th, 11th, 12th dorsal and 1st lumbar, and the 3rd sacral at the back of the thigh.

Next morning, pain had come on over the last lumbar and upper sacral spines, shooting round the crest of the ilium on both sides to the lower part of the abdomen. Towards evening these pains were joined by pain over the lower part of the sacrum and coccyx, "shooting through the privates" to the mons veneris in front. I made a note at the time, "os dilating well," and I found that manual dilatation of the os caused increase of pain over the lower sacral area. By

the night, these lower pains had got the upper hand, and practically all the pain was situated over the lower part of the sacrum and coccyx and in the perineum.

"After pains" are caused by the uterus expelling clots, &c., that have been left behind at the time of labour; for they seldom occur if the uterus is well and carefully kneaded after the expulsion of the placenta. The pains are increased at the time by the administration of ergot. They are rythmical, coming on and passing off again, and each is mostly accompanied by the passage of a clot. They are frequently accompanied by considerable tenderness which is cutaneous and follows the areas laid down in this paper.

Case 44.—Mrs. L. U. C. H., Midwifery Department.

Second stage of labour. Membranes ruptured. Os dilated. Child presenting by vertex.

Pains round the body "like a hoop." Only in the groin and back. Very marked cutaneous tenderness as in fig. 37 (right half of each diagram). After delivery the pain and tenderness began to extend down the outer side of the thigh, and almost to the knee on the inner side of thigh.

Confined 48 hours. Complains of bad after pains. Marked cutaneous tenderness as in fig. 37 (left half of each diagram).

Third day after delivery all pain gone. Slight cutaneous tenderness still remains.

Recovered as usual, and was up on the tenth day.

Fig. 37 shows the cutaneous tenderness associated with the pain of the second stage of labour on the right side, and the tenderness found forty-eight hours after labour in association with "after pains" in the same woman. It will be seen that the pain of labour is associated with tenderness over the eleventh dorsal, and the "after pains" with tenderness over the tenth, eleventh and twelfth dorsal, first lumbar, and third sacral areas.

In multiparæ, the os is frequently extensively lacerated by previous labours so that the cervical canal practically does not exist. In such cases the lower or sacral areas of pain do not seem to appear after labour. In the case of a woman whose os was deeply lacerated by the birth of a large child the year before, "after pains" associated with cutaneous tenderness only appeared over the areas corresponding to the tenth, eleventh, and twelfth dorsal areas. There was no sign of reference to the sacral areas on the back of the thigh. She said that at all her labours before she was obliged to have someone to "rub her back" (over

the lower part of the sacrum), but this time "she did not know herself," for she had no pain over any part of the back excepting where she "now has them."

It might perhaps be objected, that the few instances I have given of tenderness in the skin associated with after pains were open to doubt in that women were notoriously "hysterical." I think this objection is set aside by the fact, that out of 178 women whom I visited within 48 hours of labour, 52 had such well marked cutaneous tenderness, that I could map it out on a chart. Thus, in my experience of hospital patients delivered by students, 30 per cent. showed some cutaneous tenderness within the first 48 hours after labour. Moreover, my impression is that this tenderness is always worst in those cases in which the child was born before the arrival of the doctor, and in those cases in which the student was inexperienced (at the beginning of the month of duty), showing the relation of the pain and tenderness to incomplete evacuation of the uterus.

Thus the uterus like the bladder causes referred pain and cutaneous tenderness over a double set of areas. Anything which stretches the lower segment and especially the cervical canal proper causes pain and tenderness over the fourth, third and sometimes the second, and even first sacral areas.

On the other hand painful contractions of the uterus are accompanied by pain over the eleventh, twelfth and sometimes the tenth dorsal areas, together with occasionally the area of the first lumbar segment.

Pleura and Peritoneum.

Referred pain such as we have considered in this chapter is characterised by the following features.

1st. It tends to follow the lines of spinal segmentation rather than those of peripheral nerves. Thus on the chest it cuts across several ribs and does not follow the lines of any one intercostal space.

2nd. It tends to form bands round the body and thus in diseases of the abdominal viscera the pain may be quite as marked in the back as over the abdomen.

3rd. It is mostly associated with cutaneous tenderness, which may be quite as marked at a distance from the affected organ as in its neighbourhood. Thus gall stones are not only associated with tenderness over the area of the gall bladder in front, but also with tenderness over the ribs and spine behind. In the same way intestinal disorders may cause a band of tenderness extending round the body, not only in the loin but also overlapping the summit of the iliac crest on its way to the front.

Now I was long ago struck by the fact that in affections of the peritoneum the pain and tenderness differed fundamentally from that of referred pain, in that, although the pain is well localised, it does not follow the line of the nerve areas. Thus, for instance the patient will refer the pain to an area both above and below the umbilicus without at the same time complaining of any pain in the back or loin.

Moreover, there is no true cutaneous tenderness in peritonitis, and the tenderness that is present differs fundamentally in character from that which accompanies referred pain. If the skin only is picked up between the finger and thumb in a case of acute peritonitis the patient complains of no pain, but directly the deeper structures of the abdominal wall are included in the gentle pressure he complains of intense pain. Thus firm steady pressure as a rule relieves referred abdominal tenderness, whilst light pressure between the finger and thumb, or even the pressure of the clothes, at once elicits it. On the other hand in peritonitis light cutaneous stimulation causes no pain, but the slighest attempt at deep pressure, or even the lightest percussion, at once causes marked pain.

Again, the distribution of the two forms of tenderness differ fundamentally. The tenderness of peritonitis may be present all over the abdomen from the ensiform cartilage to the pubes and from one costal margin to the other, but it never appears at the angle of the scapula or over the sacrum and iliac crest. On the other hand, if referred tenderness extended over the area just mentioned, spots of tenderness would appear to a certainty over the ribs in the line of the

angle of the scapula, over the costal margin in the eighth and ninth spaces, over the twelfth dorsal spine, over the twelfth rib, and over the last lumbar and first sacral spines. Thus the tenderness of peritonitis only makes its appearance over those points where pressure, &c., can actually affect the peritoneum, whilst referred abdominal tenderness is always present over bony points, where pressure cannot possibly produce any effect on the abdominal cavity or its contents.

Thirdly, we find that however great may be the pain caused by the peritonitis, the abdominal reflexes are not exaggerated as would have been the case if the tenderness had been cutaneous.

Case 45.—Amelia T., aged 42, married, U. C. H. (Mr. Beck). Was admitted November 27, for obstruction of the bowels. Has never had any obstruction before, but for fourteen years has suffered from chronic constipation. Bowels open twice a week with medicine only.

November 20. Bowels last opened.

November 21. Sudden pain in lower part of abdomen, chiefly on the left side.

November 22. Went to work feeling ill.

November 24. Took medicine after which she vomited.

November 27. Admitted. Has not passed flatus or had desire to pass a motion since November 20. Had not vomited except after the purgative. Abdomen soft, but distended rather more on left than right side. Whole abdomen tympanitic to percussion.

Now it was a very striking feature, that although there was very marked tenderness of the left iliac fossa on even the lightest percussion or palpation all cutaneous tenderness was completely absent. There was no pain or tenderness in the back or loin, and the most posterior limit of the tenderness was a line from the tip of the eleventh rib to the crest of the ileum. Thus all tenderness was deep, and it was only present over those points where the peritoneal cavity could be disturbed by the manipulation.

It was thought to be a case of volvulus of the sigmoid flexure of the colon, and Mr. Beck operated at once. The colon and gut looked healthy, but patches of lymph could be seen on the coils of small intestine, and on separating these a little thin pus escaped. On portions of the small intestine were well defined patches of lymph, and it was evident that these coils had formed the lining wall of an abscess cavity. This abscess reached down into the pelvis and was found to be connected with the left Fallopian tube. Vermiform appendix was normal.

Abscess cavity was drained and the woman did well.

I now give a case of peritonitis in connection with perforation of the vermiform appendix to illustrate the same point.

Case 46.—Mary Ann B., aged 49, married. U.C.H. (Mr. Barker), Dec. 5.

November 26. Pain in abdomen began, but she did not stop work. December 3. Became so severe she went to bed. Pills caused action of bowels.

She has long suffered from chronic constipation.

December 5. Admitted. Well nourished. Tongue furred. No vomiting. Pain after taking solid food. Abdomen not much distended. Nothing definite in either iliac fossa. No difficulty or pain on micturition. Nothing definite felt per vaginam.

Great tenderness and pain in the abdomen. The pain is situated in the right iliac fossa, and the tenderness extends from the upper part of the fossa to the costal margin above. It does not extend over the middle line or further back than a line joining the tip of the eleventh rib and the crest of the ileum. There is no pain or tenderness in the loin. The most marked feature is the complete absence of cutaneous tenderness. Thus the skin can be picked up between the fingers without giving her any pain, but palpation or light percussion cause considerable pain. Abdominal reflex not obtained.

December 10. She began to vomit. Mr. Barker operated and found a small abscess in connection with the vermiform appendix.

The appendix was perforated, but was not swollen, and except for the perforation was healthy.

December 16. Died. *Post-mortem.*—No general peritonitis Abscess cavity shut in by firm adhesions, lower end of abscess just below brim of pelvis.

In contradistinction to these two cases I give two cases of strangulated hernia in which cutaneous tenderness over the tenth dorsal area was very marked. Both cases showed well the tenderness in the loin which is present when the whole bandlike area of the tenth dorsal is present. The second case developed peritonitis after enterectomy, and yet tenderness did not re-appear after the operation.

Case 47.—Hortense D., aged 68, married. U.C.H. (Mr. Beck).

For six months has noticed a small lump in right groin.

January 6. In the morning noticed that the lump was larger than usual, and was painful. Enema, which she thinks brought away fœcal matter.

January 7. Vomited six or eight times. Admitted at noon.

Abdomen distended. Walls thin. In right groin slightly oval swelling, size of pigeon's egg. Obviously a femoral hernia.

There is no deep tenderness, but definite cutaneous tenderness marking out fairly the area of the tenth dorsal segment on the right side. Abdominal reflex on right side a little brisker than on the left side.

The usual operation was performed and patient did well.

Case 48.—Elizabeth W., aged 30. U.C.H. (Mr. Barker).

Six months ago first noticed a swelling in the right groin, which disappeared with rest.

Six weeks ago rupture came down again, but went back under rest.

December 22. Rupture came down again.

December 23. Began to vomit. Bowels opened naturally this morning for the last time.

December 24. Worse. Vomiting continued.

December 27. Vomited last at 6 a.m. Admitted to U. C. H. General condition on admission very satisfactory. Obvious femoral hernia in right groin. Abdomen not distended. No tenderness on percussion or palpation. Very definite cutaneous tenderness on taking up the skin of the abdomen between the fingers. This cutaneous tenderness marked out very beautifully the tenth dorsal area on the right side, and was so definite, that it could be demonstrated several times in succession to onlookers. There was no cutaneous tenderness over the hernia itself. Thus all the tenderness was at a distance from the affected part. The superficial reflexes over the tender area were distinctly exaggerated and the head of a pin felt like a point.

She refers her pain to a point just to the right, and a little below the umbilicus (*i.e.*, the maximum of the tenth dorsal area in front). She also has some pain in the loin and back.

Mr. Barker operated, and found that the knuckle of gut in the hernia had ruptured in two places, allowing a small quantity of fœcal matter to escape. He excised a portion of gut, and sutured the two ends.

December 31. She passed flatus freely, and her bowels were opened. Seemed to be doing well.

January 4. Complained of pain in the wound and right hypochondrium, but there was no cutaneous tenderness.

Temperature began to run up for the first time, and she died in the night.

Post-mortem.—The juncture of the two ends of gut had leaked and there was general peritonitis.

Now an exactly similar law holds good for the pleura. Every physician is familiar with the numerous cases of "pain in the side" increased by breathing and coughing which are unaccompanied by the physical signs of pleurisy. Such pain is mostly associated with cutaneous tenderness and is in reality referred. In order to render clear the

difference between the pain of pleurisy and this referred pain let us consider the following example.

A man of 36 complained of cough accompanied by intense pain over an area the size of the palm of the hand situated over the sixth, seventh and eighth ribs. It extended from the level of the posterior axillary fold forwards nearly to the costal margin on the left side. There was no tenderness when the skin was lightly picked up between the finger and thumb but the most exquisite tenderness on the lighter percussion or pressure with the fingers. There was no tenderness in the abdomen or in the line of the angle of the scapula and over the vertebral spines. Over this painful area very marked pleural friction was audible. Now if this pain and tenderness had been referred it would have corresponded to the maximum of the eighth dorsal area. But with such exquisite pain the posterior and also the abdominal portion of this area must have been affected, and true cutaneous tenderness would have certainly manifested itself. I have more than once had the opportunity of seeing true referred pain accompanied by cutaneous tenderness on the one side associated with the local pains and deep tenderness of pleurisy on the other.

Another feature of the pain of pleurisy which I wish to emphasize is its conduction along the line of the intercostal nerves as opposed to those of the segmental areas. Thus a pleuritic pain situated in the ninth and tenth space in the line of the angle of the scapula will radiate forwards along these inter-spaces to about the umbilicus. From before backwards it will cut across the lines of the seventh, eighth and ninth segmental areas and will finally enter the distribution of the tenth dorsal segment. Although it radiates on to the abdomen all tenderness ceases as soon as the costal border is passed.

Case, 49.—James P., aged 56. U. C. H. (Dr. Roberts), 1892. Was at work and appeared healthy until two weeks before admission, when he developed a bad cough. The day before admission he coughed up blood, and when admitted, was still bringing up very highly blood-stained sputum.

Constant hard cough. Cough caused pain over an area the size of the palm of the hand, situated over the sixth, seventh, and

eighth ribs. It extended from the level of the posterior axillary fold forwards nearly to the costal margin. There was no superficial or cutaneous tenderness, but even light percussion or pressure with the fingers caused pain. No tenderness in the abdomen in front of the costal margin or at the angle of the scapula, and over the vertebral spines.

Left base moves less than right. Over left base in the axillary line from the fifth rib downwards, very marked pleural friction. Slight signs of consolidation at left apex and base.

This pain was removed by strapping the left side.

Thus in conclusion I think we can formulate the following statement. Affections of the serous cavities of the body do not cause referred pain or cutaneous tenderness, but produce local pain which follows the lines of peripheral nerves and is associated with deep tenderness over the affected point only.

CHAPTER IV.

THE SENSORY FIBRES IN THE SYMPATHETIC SYSTEM.

I have gone through all the organs systematically in the last chapter and attempted to show that the pain and tenderness caused by certain disturbances in their normal activity is referred along the distribution of certain somatic nerves. Now on Ross's[1] hypothesis a knowledge of the nerve roots along which reference takes place enables us to map out the sensory supply which the affected organs receive by means of the sympathetic. For on this hypothesis the viscera receive their sensory fibres from that segment of the spinal cord from which the somatic sensory roots arise along which the pain is referred.

I will therefore put together the results arrived at in the previous chapter in the form of a table. But I must first call attention again to the fact that certain organs have a tendency to refer into the supply of the cervical plexus and that of the cranial nerves. The organs in which this feature is more marked are the lungs, heart, liver, intestines, and stomach. I cannot now enter into a full discussion of the significance of this fact, but must reserve it for my next

[1] BRAIN, January, 1888.

communication in which I hope to describe the areas of referred pain and tenderness for the head and neck. I must however, for the purpose of this table state that I consider this reference to the head and neck to represent the supply of the vagus and depressor, and thus in all those cases where the disturbance of an organ produces direct reference above the clavicles I have inserted these nerves in my tabular statement of the segments from which they are supplied.

Heart.—1st, 2nd, 3rd, dorsal segments.
Cervical plexus [= depressor ?]

Lungs.—1st, 2nd, 3rd, 4th, 5th dorsal.
Cervical plexus [= vagus ?]

Stomach.—6th, 7th, 8th, 9th dorsal.
Cardiac end from 6th and 7th.
Pyloric end from 9th.

Intestines.— [A.] Down to upper part of rectum.
9th, 10th, 11th and 12th dorsal.

[B.] Rectum.
2nd, 3rd and 4th sacral.

Liver and *Gall Bladder.*—7th, 8th, 9th, 10th dorsal.
Perhaps 6th dorsal.
Cervical plexus [= vagus ?]

Kidney and *Ureter.*—10th, 11th and 12th. The nearer the lesion lies to the kidney the more is the pain and tenderness associated with the 10th dorsal. The lower the lesion in the ureter the more does the 1st lumbar tend to appear.

Bladder.— [A.] Mucous membrane and neck of bladder.
[1st.] 2nd, 3rd, 4th sacral.

[B.] Over-distension and ineffectual contraction.
11th and 12th dorsal and 1st lumbar.

Prostate.—10th, 11th, [12th] dorsal.
1st, 2nd, 3rd sacral and 5th lumbar.

Epididymis.—11th and 12th dorsal and 1st lumbar.
Testis.—10th dorsal.

Ovary.—10th dorsal.

Appendages, &c.—11th and 12th dorsal and 1st lumbar.

Uterus.— [A.] In contraction,
10th, 11th, 12th dorsal and 1st lumbar.
[B.] Os uteri,
[1st], 2nd, 3rd, 4th sacral [and 5th lumbar very rarely].

It is obvious from this table that the sensory supply of the viscera bears a very curious resemblance to Gaskell's[1] scheme of their motor and inhibitory supply. I was therefore exceedingly interested to hear from Dr. Gaskell that my supply closely represented the results obtained by Edgeworth by anatomical methods. Edgeworth's paper in the May number of the *Journal of Physiology* showed how closely we were agreed and yet how different were our results on certain points. Firstly as to the differences, which I do not think are essential. Edgeworth dissected and examined microscopically the sympathetic branches in the dog. Now this animal possesses thirteen dorsal and seven lumbar roots. Edgeworth finds that the large medullated fibres which he believes to be the sensory fibres of the viscera come off from the first dorsal to the third lumbar roots inclusive. There is then a blank of three roots from which no such fibres come off followed by another outburst of these fibres from the region of the seventh lumbar and first and second sacral. Now in man there are but twelve dorsal and five lumbar vertebræ and I find that my method gives a supply of visceral sensory fibres from the first dorsal to the first lumbar segments with perhaps also very rarely the second lumbar. Then follows a blank of three roots followed by a lumbo-sacral outburst from the fifth lumbar and first, second, third and fourth sacral. Of these the fifth lumbar is only occasionally seen. Now I think that considering the different anatomical construction of man and the dog these differences are but of slight importance. And now to pass on to the points upon which we arrived at similar results. Edgeworth finds that the heart, lungs, stomach, liver, and intestines

[1] *Journal of Physiology*, vol. vii., 1885.

are all supplied by large fibres from above which reach them in the vagus, and in the case of the heart by the depressor nerve. Now these are the organs which I believe give reference into the areas above the clavicle, *i.e.*, into the region of the head and neck above the upper gap. The most definite reference into the neck is given by the liver, lungs, and heart.

Another point on which we are united as will be seen by comparing my table with Edgeworth's chart is in the double supply of the bladder. The double supply of the prostate and uterus he does not seem to mention. Thus I think that the results produced by both the anatomical (Edgeworth) and by the physiological methods tend to show that the sensory distribution of the sympathetic essentially follows the lines laid down by Gaskell for the course of its motor and inhibitory fibres.

Before I leave this subject I should like to mention some interesting points brought out by the recognition of the double supply of the bladder. In those cases where the distribution of the analgesia points to a lesion of the sacral region of the spinal cord the patient says he knows when he wants to pass water but he has no power of retaining it. As soon as a few ounces have collected in his bladder he "feels he wants to pass it and cannot hold it back." It then comes from him involuntarily. A patient in this condition has analgesia over the areas corresponding to the third and fourth sacral segments.

If, on the other hand, the analgesia is over an area corresponding to the eleventh and twelfth dorsal and first lumbar areas, and sensation is normal over the sacral area, the condition of the bladder is exactly the reverse. The patient is quite unconscious of the desire to pass water, and the bladder will gradually fill until it contains 40 to 60 ounces of fluid. If a catheter is not passed the urine will gradually accumulate until it begins to dribble past the sphincter from mechanical pressure, and the condition known as "dribbling from overflow" will have been established.

If, however, the distribution of the analgesia points to a local lesion above the ninth dorsal segment, a third condition

of the bladder will appear. The urine will collect in the bladder until it has reached 15 to 20 ounces, and then will be passed without the patient's knowledge. Thus here the passage of urine is a normal but completely automatic act.

In the first class of cases the bed is always wet; in the second class the bed is never wetted if a catheter is passed about three times in the twenty-four hours; in the third class the bed is wetted three or four times only in the twenty-four hours, and is then saturated with a large quantity of urine.

Throughout the study of cases of nervous disease, evidence of the relation between pathological condition of certain viscera and sensory disturbances in the superficial structures of the body is constantly manifested. For instance, a man with caries of the spine suffered from a girdle sensation round the area of the eighth dorsal segment. At the same time he was greatly troubled by flatulent dyspepsia, which was untouched by drugs. It was, however, greatly relieved by counter irritation applied to the maximum tender point of the eighth dorsal area in the eighth space and mid axillary line.

Again in a case of tabes with analgesia over the area supplied by the tenth dorsal root, I saw an attack of complete paralysis of the small intestines simulating intestinal obstruction.

Thus, in conclusion, I feel inclined to think that the sensory supply of the viscera agrees with Dr. Gaskell's scheme for their motor and inhibitory supply in the following points:—

[1st]. The sensory fibres enter the central nervous system in three great groups. The highest of these is in the head and neck, the middle group lies beween the 1st dorsal and 1st lumbar segments of the spinal cord; the lowest group extends from the 5th lumbar to the 4th sacral segments.

[2nd]. Thus there are two gaps in the spinal cord which are not in connection with sensory fibres from the viscera. The higher of these consists of the 5th, 6th, 7th and 8th cervical segments, whilst the lower corresponds to the 2nd, 3rd and 4th lumbar segments.

CHAPTER V.

On "Generalisation" and General Diseases.

So far, I have spoken as if disturbances in the activity of one organ produced pain and tenderness over certain definite areas only. If the human organism is otherwise perfectly healthy, this is so; but after the pain has remained localised for a considerable time, the condition of the central nervous system becomes profoundly altered. I pointed out above that in biliary colic, the tenderness over the eighth dorsal area generally makes its appearance on the left side as well as on the right. When one testicle is affected, the tenderness also tends to be bilateral, and the same applies to the kidneys, especially if the disease is a long and wearing one. Thus, there is a great tendency for both pain and tenderness to spread to the opposite side at the same spinal level. In all these cases it might be suggested that the organ receives most of its fibres from one side, but also a certain number from the other side of the cord; and there is probably much truth in this supposition.

But there is another and much more important form of diffusion or spreading. If I have an aching tooth, the pain is at first localised to the tooth affected. The longer the toothache continues, the more I become worn out, and the pain is rapidly accentuated by a "neuralgia," *i.e.*, that is a pain in the face. This neuralgia is soon accompanied by distinct cutaneous tenderness over a definite area on the face corresponding to the tooth affected. If I am anæmic, or if the pain remains untreated until my bodily health is affected, I no longer have a localised area of tenderness, but the pain and with it the tenderness spreads until the whole of one-half of the head and even the neck may be intensely tender. Thus at last the pain of an aching tooth has produced tenderness over areas which bear no relation to the affected organ.

But there is another method by which the pain caused by one organ may be ultimately accompanied by pain over an area corresponding to another organ. Certain viscera such as the uterus and breast are naturally closely related to

one another. Thus suppose pain and tenderness to exist in consequence of the disease of one of these organs, the next area that will be affected when this pain and tenderness spreads, is that belonging to the associated organ. Thus within twelve hours after labour, it is not uncommon to find a woman suffering from a pain under the heart associated with cutaneous tenderness which comes and goes with the "after pains." This pain under the heart may be increased by ergot, or by manipulating the uterus.

With orchitis or prostatitis, we also occasionally find that the patient complains of a pain at the epigastrium, representing the stomach area. This is put down to "hypochondriasis," and if it occurs in a woman as a consequence of ovaritis, she is said to be "hysterical." But this phenomenon is no more "hysterical," whatever that may mean, than is the reference of the pain and tenderness of an aching tooth to the back of the head or the shoulder.

This is the phenomenon which I have been accustomed to call "generalisation" of visceral pain and tenderness, and is of such common occurrence as to form a very important factor in the clinical picture of many diseases.

The order in which generalisation takes place, leads one to speak of the relative "specific resistance" of the centres for the sensory impulses from various organs. No very definite rule can be laid down to govern every case, but each case must be considered on its merits. However, the area which appears most easily in a woman, as a secondary affection, is the tenth dorsal; then perhaps the sixth dorsal, or inframammary, and then the various gastric areas, beginning first with the ensiform or seventh dorsal. In a man, the tenth dorsal appears rather less readily, whilst the ensiform appears with great ease. The cervical areas appear with ease in both sexes. On the other hand, the third and fourth dorsal associated with the lung, rarely appear as secondary affections. Thus the central connections of these areas in the cord appear to have a different specific resistance; some of them appear with ease even when the organs with which they are primarily associated are not affected, whilst others only appear after every other area in the body has become tender.

Now the greatest specific resistance seems to be possessed by the areas of the two gaps; and when the gaps become affected, it is only by a gradual creeping onward of the neighbouring areas. Thus, if we watch the genesis of universal tenderness in the leg, we find that first of all the twelfth dorsal and first lumbar in front, and the first, second, sacral and fifth lumbar of the posterior group are all tender. Then from the first lumbar above, the tenderness creeps over the second lumbar, whilst a similar process is occurring in the fourth lumbar below. At first, only the tender points appear, and then the areas gradually develop until the gap is filled up, and the whole leg is now tender.

In this connection I may say a few words on the difficult subject of dysmenorrhœa. A certain number of cases of dysmenorrhœa are entirely free from pain and tenderness in the inter-menstrual period, but develop definitely localised pain and tenderness, following the lines I have laid down, during menstruation. Of this class, the most satisfactory are those with some affection of the cervical canal, such as chronic cervical catarrh or polypi. They may be entirely free from pain until the flow comes on, when they develop pain and tenderness over one or more of the following areas: —the eleventh or twelfth dorsal, first lumbar, fourth, third, or second sacral. In some cases a considerable number of these areas are affected, but even at the height of the disturbance the tenderness does not transgress the limits of the areas supplying the pelvic viscera. Moreover, the areas corresponding to the part which is chronically diseased are always most affected.

With slight degrees of ovaritis, a second type of dysmenorrhœa makes its appearance. Here there is generally pain and some tenderness, especially after examination, over the tenth dorsal area, in the intermenstrual period. Just before menstruation begins, this tenderness becomes very intense, and by the time the flow is established all the areas which correspond to the pelvic organs are affected. Moreover, the areas of the gap may become tender by extension, until at last the whole body and leg, below the upper border of the tenth dorsal area, may be tender. In such cases, not

only are the cutaneous (superficial) reflexes exaggerated, but the knee-jerk also becomes exaggerated. This is beautifully seen in those rare cases where the tenderness remains unilateral.

Now we pass to the third class of pain during menstruation in which the patient has some local affection which causes more or less constant visceral pain. This may be some error of refraction which causes its proper tenderness (which I shall describe and discuss in my next paper) over the middle of the upper lid and front of the scalp. It may be chronic cough, causing tenderness over the maxima of the second dorsal. It may be chronic gastric disturbance, causing pain over one of the gastric areas; or it may be chronic constipation, producing the well-known pains over the tenth and eleventh dorsal areas. Whatever form this pain normally assumes, it is enormously increased by the disturbing influence of menstruation on the nervous system. The hypermetrope will suffer from agonising headache, which may spread on to the neck. The patient with the cough suffers from pains and tenderness in the chest and arms, and in the lower branches of the cervical plexus. The patient with gastric disturbance has tenderness over more or less every gastric area. Moreover, there may be two centres of spread, which gradually widen until they meet, and thus there may be hardly a part of the body that is not tender. And now the patient is put down as "neurotic" or "hysterical," and all thought of rational treatment abandoned.

But as I shall show below the same phenomenon appears in even the most stolid of us males under the influence of a rise of temperature. Anyone who has suffered from a bad tonsillitis with fever, knows to a certain extent the symptoms of dysmenorrhœa, with its pain, aching and tenderness in the loins and back.

Thus the changes in the nervous system accompanying menstruation must be put down as one of the most potent causes of "generalisation" of visceral pain and tenderness.

Again, as I pointed out above (p. 92) a normal labour is frequently followed by some pain and cutaneous tenderness.

But the pain and tenderness very rarely overstep the areas associated with the pelvic viscera. But after abortion "generalisation" is very common, and although the course of the puerperium is normal, the patient will frequently complain of pain and tenderness elsewhere than over areas representing the pelvic viscera. It would appear as if the resistance of the nervous system were increased *pari passu* with the progress of normal pregnancy to bear the strain of normal labour; but that when abortion takes place it is unprepared for the strain put upon it and generalisation takes place easily.

Anæmia is one of the commonest causes of such generalisation. A patient may be anæmic and yet suffer from no pain or tenderness. But as soon as some cause for referred pain is present generalisation at once begins. Thus if an anæmic women menstruates she not only has pain over the tenth dorsal area corresponding to the ovary, but she has pain and tenderness over the areas of the sixth dorsal (heart), seventh dorsal (stomach), occipital and midorbital (eyes, etc.) If an anæmic man or woman have a toothache or suffer from hypermetropia they may have the most widespread tenderness and pain. And yet if the cause is removed this pain does not cease, at any rate immediately, for the resistance of the centre being once broken down is with difficulty recovered. In every such case of anæmia I believe the starting point for generalisation will be found in the affection of some organ. Where widespread tenderness of the head and neck are present and headache is a marked feature, some error of refraction is often present. In some cases the starting point of the pain is in the cough of some lung or bronchial affection. A very large number start originally in the pain of chronic dyspepsia, and an even larger number in the pain caused by some irregularity in the pelvic viscera.

Thus it is not sufficient simply to treat the anæmia or to treat the original cause and point of primary manifestation of pain, but both must be corrected.

But anæmia is not the only agent which will so lower the specific resistance of the central nervous system that a local

pain will generalize. A sudden rise of temperature will produce the same effect. Follicular tonsillitis or any parenchymatous affection of the tonsils is associated with cutaneous tenderness, extending round the neck from the lower part of the mastoid behind, almost to the middle line of the neck just above the pomum adami. If one tonsil only is affected one side of the neck alone is tender over this area. But let the onset be sudden and associated with a rise of temperature to 101° or 102° F. We shall now find the same zone of tenderness, but in addition there will be pain and tenderness over the forehead, in the occipital region, over the maximum of the tenth dorsal area in the back, and at the epigastrium (representing the seventh dorsal), and probably also at several other points in the body and limbs, all of which represent the maxima of their various segmental areas. Thus a rise of temperature has added, to a simple area of tenderness representing the organ primarily affected, a multiplicity of painful and tender spots representing areas which could in no way be effected by the local manifestation of the disease.

I have attempted to show how a sudden rise of temperature may convert a simple patch of referred tenderness into a wide spread affection by breaking down the resistance in the central nervous system, which normally resist generalisation. And in some cases a sudden rise of temperature will produce this wide spread tenderness without our being able to recognise any area of primary disturbance. This condition is produced most easily by true epidemic influenza. In this disease the patient notoriously complains of "pains all over," and careful examination shows that these pains are associated with tenderness in the skin. A glance at fig. 38 is sufficient to reveal the resemblance of these points of cutaneous tenderness to the "maxima" of which I have spoken so frequently in this paper. In those cases of influenza which have their greatest incidence on the lungs, the most prominent spots are situated over those areas which usually appear in pulmonary disturbances. In the intestinal group, on the other hand, the maximum disturbance falls over the spots related to the intestinal tract. In some cases of influenza where the local manifestations in any

one organ were very marked, I have actually seen whole spinal areas marked out by tenderness. In every case I have selected male cases and only such as had a well marked elevation of temperature, making the diagnosis a certainty. Moreover, I have only taken cases which occurred during the last epidemic, and have not included sporadic cases occurring at other times of the year.

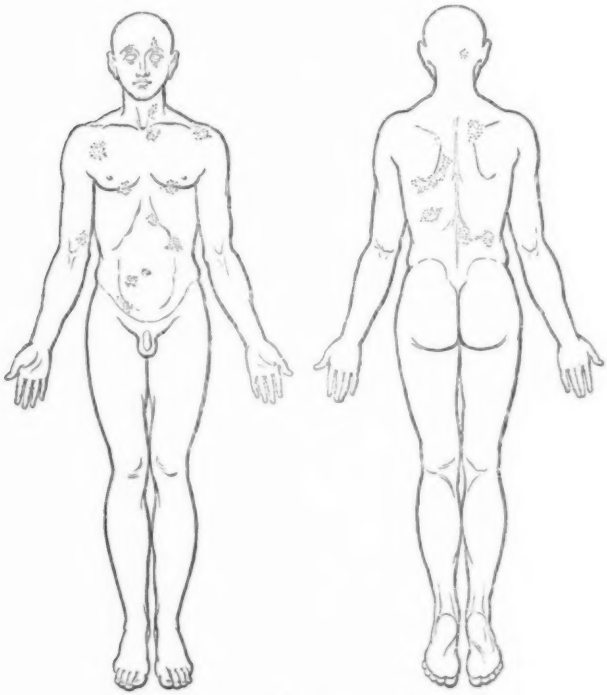


FIG. 38.

On the right hand side of the body in each figure are shown the areas of cutaneous tenderness during an attack of influenza in Case No. 50.

On the left hand side of the body are shown the areas of cutaneous tenderness which appeared during an attack of influenza in Case No. 51.

Case 50.—Robert F., aged 35. U. C. H., January 4, 1892.

January 1. Cold shivers, headache, pains all over especially in the joints.

January 4. Temperature 100° F.

Great cutaneous tenderness over the areas on the right hand side of fig. 38. His eyes ache and water, and the whole upper part of the body feels as if it had been "bashed."

Case 51.—Frank B., aged 16. U. C. H., January 6, 1892.

Typical influenza. Temperature 103°. Pains in head, eyes and upper part of body.

Marked cutaneous tenderness as on left hand side of fig. 38.

But influenza is not the only specific disease which produces these tender areas; they are sometimes present in typhoid fever. The most marked case I have ever seen was in my own brother in whom the temperature ran up very rapidly reaching 102° on the second day and 104° on the third day. He was very bright in spite of the high temperature and was able to give me a brilliant description of the locality of his pains and the peculiar "beaten" feeling he had over certain spots of his body. Fig. 39 shows the tender areas on the third day. As he lay in bed he tested his own body and without my suggestion found the greater number of the tender areas himself and marked them on his body for me. Such a condition is however uncommon in typhoid, for the temperature does not run up as a rule rapidly enough to produce this wide spread tenderness. The usual condition of a patient suffering from typhoid in the first week is a band of tenderness more or less well marked over the area of the tenth or eleventh dorsal segments with spots over the forehead corresponding to areas I shall discuss in my next paper. I have however seen one case besides the one just mentioned in which the tenderness was fairly wide spread and in this case also the temperature ran up rather rapidly.

Case 52.—Christopher H., aged 22.

Seized with frontal headache on night of December 22, 1891.

On evening of December 23, temp. 102°. He complained of headache over the eyes and pain in the right shoulder when he worked his Indian clubs. He also had pain in the lumbar region when he stooped.

Dec. 24. Temp. reached 104°. Cutaneous tenderness over his forehead associated with frontal headache. Cutaneous tenderness over the abdomen and loins marking out the tenth dorsal spinal area. The tenderness is so marked that he called a touch with the blunt end of the pin "a prick."

Dec. 25. Temp. now varies between 102° and 103.8°. The tender band in the abdomen and loin is no longer so well marked, but the whole body and head are dotted over with points of cutaneous tenderness. He himself found cutaneous tenderness over the following points and drew my attention to them. Tip of ninth rib, upper and outer part of thigh, angle of scapula, abdomen and loin, and in the spot on the calf. He said sometimes his back was worse, sometimes his calf, and sometimes his head.

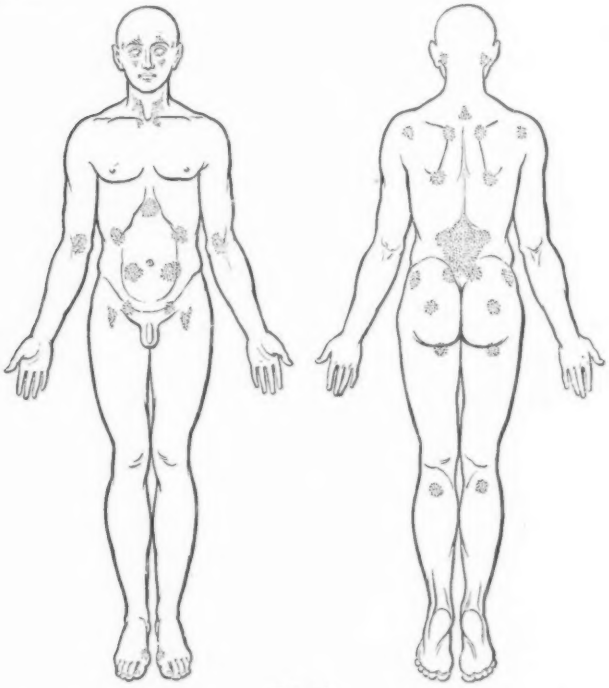


FIG. 39.

To show the areas of cutaneous tenderness which appeared in a case of typhoid in which the temperature ran up rapidly (Case No. 52).

Dec. 28. All cutaneous tenderness has disappeared. The temperature now varies between 102.4° and 103.6°. He has one stool daily and vomits slightly between midnight and 3 a.m.

Dec. 31. Typical diarrhœa set in and he is now beginning to get some cutaneous tenderness over the ninth and tenth spinal areas. The spleen can just be felt.

Jan. 9. The temperature is beginning to fall. Diarrhœa is ceasing.

Jan. 16. Temp. has not risen above normal. Uninterrupted convalescence without complication.

Thus this case showed a typical attack of severe uncomplicated typhoid differing from those seen every day in the rapid rise of temperature and in the fact that observations were made from the second day of the illness.

In acute rheumatism in the very early stages before the joints begin to swell definitely, similar cutaneous tenderness may also appear, though seldom, and always to a less extent than in the diseases I have mentioned so far. I saw one case of a man in whom the cutaneous tenderness was so marked that he was sent into the ward as a case of "hysterical" disease with rise of temperature. The next morning the joints had swollen and the cutaneous tenderness in the "ovarian" and "supra-orbital" regions had gone.

In one case of smallpox which I was fortunate enough to see in the stage of petechial rash not only was there tenderness (*vide* fig. 40) but the rash marked out certain areas in a beautiful manner; and I then saw that the so-called femoral triangles which are said to be so characteristic of this rash are in reality a rash marking out the thigh flaps of the twelfth dorsal spinal area. However the distribution of rashes from the point of view of the areas I have described in this paper are too big a subject to treat here.

Case 53.—John M. aged 18. U.C.H.

Has been ill nearly 8 days. The typical variola rash is beginning to appear irregularly all over his body. The early petechial rash is still present over the areas in fig. 40.

He looks very ill and complains of pain in the back (lumbar region) and in the abdomen. Definite cutaneous tenderness (head of pin called "prick") over the areas shown on the left hand side of fig. 40.

As we are discussing fevers it is interesting to notice that the drugs which are so much used to relieve referred and nervous pains are the antipyretics, such as antipyrin, antifebrin, phenacetin, &c.

In all the cases of generalisation which we have considered thus far the cause of the diminished resistance in the nervous system was a definite and recognisable one, such

as anæmia, chronic pain or pyrexia. In the next class of cases the diminution in resistance is produced by some change in the nervous system itself. This primary central break down of resistance may be an inherent defect in the nervous system of the individual or it may be produced by some psychical shock.

Thus in cases of salpingitis and dysmenorrhœa it is not at all uncommon for the pain and cutaneous tenderness to lie strictly over the areas which correspond to the organs affected until, under the influence of some mental trouble, a love affair or a death, this pain and tenderness will suddenly generalise extending to the arms, chest and head, and the

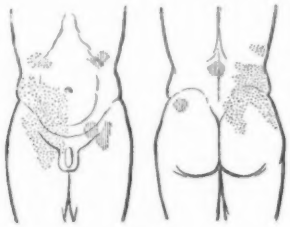


FIG. 40.

The right hand side of the body shows the distribution of the petechial rash in a case of small pox. Note the curious resemblance of the "femoral triangles," so characteristic of this rash, to the 12th dorsal area.

On the left hand side of the body is shown the cutaneous tenderness which accompanied this rash. This tenderness was associated with the well-known "aching in the back and groins."

The rash has been represented on the one side and the tenderness on the other for the sake of clearness.

case will be converted into one of "hysteria." As soon as the generalisation has taken place local measures cease to afford relief, and removal of the ovaries or tubes will only add fuel to the fire by superposing a second cause for decreased resistance.

This leads me to a consideration of the various forms of altered sensation which occur in "hysteria." I should feel inclined to divide them into two great groups.

1st. The cerebro-spinal.

2nd. The psychical type.

In the cerebro-spinal type I have been able to watch the gradual development of a case through the following stages.

(*a*) The stage in which there was well marked tenderness over areas corresponding to a diseased organ.

(*b*) Generalisation under the influence of long continued pain, anæmia or mental shock, so that the whole body becomes dotted over with areas of cutaneous tenderness.

(*c*) The cutaneous tenderness then passed into analgesia or loss of the sensation of pain with intact sense of touch.

(*d*) Touch also became affected and the greater part of the body became insensitive to pain, touch, heat or cold.

Now the characteristics of the cerebro-spinal type are as follows:—

(1) The sensory disturbance is always mainly one of pain, heat and cold, and the area affected corresponds to the areas of spinal segmentation at any rate in the early stages. (Later they may became altered by secondary suggestion). Fig. 41.

(2) The reflexes are always increased over the affected spots when the disturbance takes the form of hyperalgesia or lost when it takes the form of analgesia.

(3) There is as a rule no limitation of the visual field in the cerebro-spinal type.

On the other hand in the psychical type—

(1) Alterations follow natural lines,[1] *i.e.*, the loss of sensation runs round the limb limited above by the ankle joint, by the knee or by Poupart's ligament, loss of sensation on the abdomen occupies what is vulgarly called "the belly" or the "back," *i.e.*, it is limited by the costal margin and the lines of the iliac bones in front or by "the spine" behind. It takes "the eye" (without the forehead), "the mouth" (without the lips), "the chest" (without the arms). In fact the loss of sensation follows fundamentally different lines from that in the first class. Fig. 42.

(2) Sensation of touch, pain, heat and cold are absolutely coterminous, whereas in the first type pain is always lost before the sensation of touch and the two are lost to a very different extent.

[1] These may be ultimately found to correspond to the cortical distribution of sensation.

(3) The reflexes are unaltered over the anæsthetic or analgesic areas. This I believe to be a very valuable distinction.

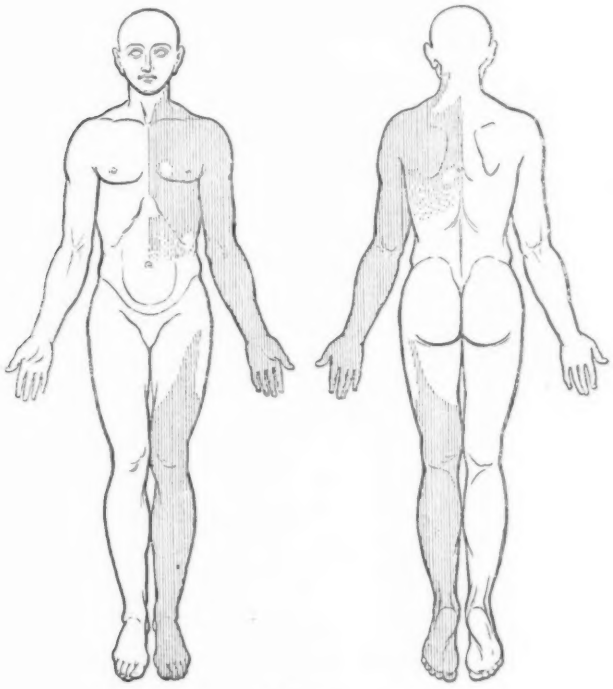


FIG. 41.

To show the distribution of analgesia in a case of "functional" disease (cerebro-spinal type) Case No. 54. All superficial reflexes to painful stimuli, and to hot and cold stimulation were lost over the areas in the figure.

The loss of sensation to touch (anæsthesia), was much less extensive, and was not co-terminous with that of pain, heat, and cold.

(4) There is almost always considerable limitation of the visual field in the psychical type (or hysteria proper).

Again the prognosis in the psychical type is much better than in the cerebro-spinal type.

Case 54.—Cissy P., aged 20, farm labourer. Queen Square (Dr. Buzzard), February, 1892.

August 1, 1891. Rose in the morning with pain in left side in mid-axilla and in front of chest under left breast; came as far forwards as base of sternum. "Like a knife" stabbing through from the front to the back, it made her cry out and feel faint. Went out to stack hurdles but was obliged to come home. Went to bed; hot skin; thirsty; no appetite.

August 4. Got up and went to work. Had to come home again on account of the pain. Went to bed and had a regular shivering fit, lasting about half an hour. Teeth chattered and shook all over. Then became very hot. Stopped at home, mostly in bed, for two weeks, and then went away for two weeks more. During the first two weeks "had an illness" (no details obtainable), but did not call in a doctor.

On returning to work in September felt the pain in the side again; but in twenty-four hours the pain disappeared, and she noticed that the left side was "numb." She could not hold the hurdle bands in her left hand and the left foot became numb and cold.

This gradually increased. In December began to walk with a stick.

Always healthy up till August, 1891. Has worked as a farm labourer for several years.

Menstruation began at sixteen. Every four weeks. Lasted five days. No pain. About November, 1891, it became irregular and has remained so ever since.

Condition on admission.—Medium sized girl. Not anæmic. Fairly intelligent. Bucolic stolidity. Sleeps well. No vomiting. No headache.

Stands well and does not stagger with the eyes shut. Drags left foot markedly as she walks. Left leg below the knee dragged like an inert mass. Grasp of right hand 40, left hand 30. All movements of left leg and foot well performed. Can dorsiflex and extend ankle.

All sense of position in left foot lost. Sense of position in left ankle impaired. Can recognise position of knee. Sense of locality in left hand perfect.

Sensation of touch impaired over the greater part of the left side of the body. Thus all sensation of touch is lost in the left leg below the knee, back and front; in the left hand, arm, shoulder and trunk, from a little above the angle of the jaw downwards, as far as a line running forwards from above the ninth dorsal vertebra to a little above the umbilicus. Neither upper nor lower limits are sharp lines, but gradually shade off. Moreover, the anæsthesia does not quite reach the middle line in front or behind. Below the umbilicus the sensation of touch is normal, but the upper and outer parts of the thigh are partially anæsthetic to touch.

The analgesia, or loss of painful sensation, is as in fig. 41 Sensation of heat and cold are lost over practically the same areas.

The superficial reflexes are absent on the left side, over the analgesic and anæsthetic areas. Thus the plantar reflex is abolished on the left side, to both touch and pain. Superficial reflexes are normal on the right side. Thus the reflexes are absent from the upper part of the abdomen on the left side, but present below the umbilicus.

Knee-jerks brisk both sides. Wrist tap present on both sides, but distinctly more ready on the right side than the left. Pharyngeal reflex present on both sides.

Vision normal. No diminution of the field of vision. No colour blindness. No ophthalmoscopic change.

Hearing normal right side. Watch heard only in contact left side.

Taste and smell good.

Loss of sensation to touch on left half of tongue.

Pain lost on a strip occupying the outer portion of the posterior part of the left half of the tongue.

No ocular paralysis. No nystagmus. Pupils equal; react to light and accommodation.

No pain or tenderness in the spine.

Urine, sp. gr. 1020; acid normal.

She rapidly improved, and on March 1, 1892, sensation was good all over body and limbs, and the superficial reflexes had returned. Discharged cured.

Case 55.—Samuel R., aged 13. Queen Square (Dr. Ramskill and Mr. Horsley).

January 6, 1892. Severe blow across left hip and lower part of spine. He went to school but was lame the same evening and the next day.

Two or three days later sharp stabbing pain came on in the lumbar region. It shot down the left leg.

Six days after the blow took to his bed on account of increasing pain. He said he could not move on account of the pain. When in bed he felt numb in the upper part of legs, thighs and hips. He had occipital headache and intolerance of light.

From January 6 to January 10 passed no urine. Also passed none from January 12 to January 16.

In April he was seen by Mr. Horsley, who sent him into Queen Square Hospital.

On admission.—Fairly well nourished boy of 13. Slight vertical headache. Fairly intelligent but lethargic. Dislikes the effort of taking part in the amusements of the ward. Could not at first be got to do the fancy work with which most of the patients amused themselves. Sleeps well, but at the onset of the illness used to dream he was falling almost every night.

Complains of slight pain in the back over the third and fourth lumber spines. Some tenderness on percussion but there is a well marked bruise in this position.

No vomiting.

He has some difficulty in raising himself in bed for he says it causes pain in the back. All movements can be performed by the lower limbs, but he complains of pain in the lower part of the back when he lifts his legs off the bed. Can stand and walk without help, but cannot walk far on account of the pain in the back.

Sensation to touch, pain, heat and cold are lost over the area marked in fig. 42. Yet in spite of this loss of sensation the cremasteric reflex can be beautifully obtained on both sides from

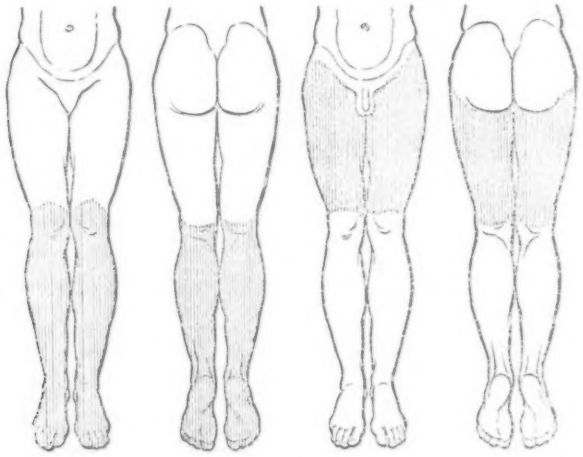


FIG. 42.

The figures on the left side represent the loss of sensation to touch, pain, heat, and cold in a "hysterical case," No. 56 (Psychical type).

The figures on the right side represent the loss of sensation to touch, pain, heat, and cold in a case of "traumatic hysteria," No. 55 (Psychical type).

the front of the thigh and the gluteal reflex from the posterior aspect. It is interesting to note that the cremasteric reflex can be obtained from the front of the thigh as far out as the level of the anterior border of the great trochanter. At this point we begin to change on to the sacral areas, and the gluteal reflex replaces the cremasteric. This experiment could be repeatedly performed in the patient in spite of the anæsthesia. Sensation over the rest of the body is normal.

Knee-jerk present and equal; not exaggerated. No ankle clonus. Plantar reflexes present. Wrist tap normal.

Smell good. Taste good. Hearing good.

Very marked limitation of the field of vision in both eyes.

No ophthalmoscopic changes.

No ocular paralysis. No nystagmus.

Pupils medium sized. React to light and accommodation.

All other cranial nerves good. Tongue protruded straight.

Appetite poor. Tongue clean. Tendency to constipation.

No physical sign in abdomen.

Heart sounds normal, no murmur.

Urine only passed two to three times a day. Quantity uncertain, sp. gr. 1020. Acid. No albumen.

He was placed on Weir Mitchell treatment.

Sensation varied a good deal. Sometimes the lower limit of the loss was at the knee. Sometimes at the ankle and sometimes only the sole of the foot was unaffected.

He increased in weight from 6st. 2¼lbs., to 6st. 8lbs., in seven weeks.

He was discharged cured October 10, 1892.

Case 56.—Mary G., nurse, single, aged 32. Queen Square (Dr. Tooth).

Summer of 1891, pain in upper lumbar region of the back.

October, 1891. Pain and tight feeling about lower part of chest. Six weeks afterwards began to vomit directly after food. She is said to have vomited blood, but never in large quantities. Was supposed to have gastric ulcer.

The vomiting continued two to three times a week until February, 1892, when she changed her doctor. The new doctor stopped the vomiting immediately.

She appears to have had influenza about Christmas, 1891, which left her with pain and tenderness in the spine, extending from the bottom of the back to the top of the head. This pain became gradually worse, and she began to slowly lose power in her legs. Was now treated for spinal caries.

No incontinence or retention of urine at any time.

Constipated. An enema has been administered three times a week ever since October, 1891.

Has always lived at home with her mother, and has never earned her living though she calls herself a nurse.

Is very liable to fainting. Very easily upset.

Menstruation regular every four weeks. Lasts three days. Not profuse. No marked pain.

Marked family history of phthisis, but not of neuroses.

Condition on admission, August 17, 1892:—

Medium sized woman of 32. Face pale. Lips somewhat pale. No œdema. Thin and ill-nourished. Temperature 98°, both in the morning and the evening.

She lies on her back complaining of pain in the spine and inability to move her legs. Quick to observe the occurrences in the ward. Is fond of reading. Memory fair. Answers questions

well. Voice low. Speech normal. Occipital and vertical headache. No vomiting for several months.

She cannot stand, and cries bitterly when she is taken out of bed, and supported on her feet. She complains that it causes her pain in about the region of the second lumbar spine. She cannot dorsiflex or extend the foot to command. If, however, the foot is passively dorsiflexed all the anterior tibial group of muscles are seen to contract. Moreover, when her leg hangs out of bed the feet remain in the normal position and do not drop. She cannot extend the foot or move the toes to command, but the foot remains in the extended position even when opposed to gravity. She cannot extend the knee to command, but when the leg is lifted off the bed the knee remains rigidly extended. Again, if the thigh is flexed passively the leg does not drop until after a considerable interval. Grasp is practically non-existent under observation, but appears to be fairly good for ordinary purposes. She does not complain of paralysis of the arms, but it is very difficult to get her to perform any movement of the arms to command.

The legs are thin and the muscles flabby, but there is no marked wasting of any group of muscles.

Electrical reactions normal.

She complains of pain in the upper lumbar region. There is intense cutaneous tenderness all down the spine limited to an area about two-and-a-half inches in breadth. This merges above into an area of tenderness over the occiput.

Touch, pain, heat, and cold are not felt over the area in fig. 42. The limits of the loss of sensation are absolutely coterminous for all forms. Sensation of rest of body normal.

She seems to have lost all sense of position as high as, and including the knee. She does not seem to know when her muscles contract under the faradic current as high as the knee. She can bear the strongest faradic current without flinching over the anæsthetic area.

Knee jerk exaggerated on both tides. Wrist tap brisk both sides.

No ankle clonus.

Plantar reflexes present to touch, but slightly diminished. Fairly brisk to a pin prick. Thus reflexes can be obtained, even over the anæsthetic area.

Abdominal reflexes good.

Pharyngeal reflex absent on both sides.

In spite of the tenderness of the spine, the usual tests for bone disease give a negative result. No visible abnormality in the spinal column.

Taste, smell, and hearing good.

Myopic. No opthalmoscopic change in fundus or discs.

When tested with Snellen's test types, says she can only read $\frac{6}{200}$ [$\frac{2}{60}$]. Again, she reads Jäger 1 at the usual distance, but rapidly approximates the board to her face, saying it gets quickly

dim. Finally, she only reads J 10 badly. This occurs with each eye.

Marked diminution in the field of vision for white and black and to a still greater extent for colours.

No nystagmus. Cranial nerves normal, excepting that the sensation of the pharynx seems to be abolished.

Sphincters normal.

Circulatory and respiratory systems are normal.

Appetite poor, tongue clean. No enema was administered during her stay in the hospital, but she was told that her bowels would act normally. They acted regularly every day without artificial aid of any kind.

August 27. Feels the wire brush, and cries with the pain.

August 29. All forms of sensation now good. They returned simultaneously.

August 30. Can only bear wire brush at half the full strength.

September 1. Moves legs to command, though slowly.

September 8. Pharyngeal reflex has returned.

December 7. She left the hospital cured, excepting for occasional pain in the back.

These three cases exemplify the difference between the two types of so-called hysteria. For Case No. 54 was characterised by the following points:—

(1) The loss of sensation followed the lines laid down in this paper for the distribution of sensory disturbances in visceral disease.

(2) Reflexes were abolished over the anæsthetic area.

(3) There was no limitation of the field of vision.

On the other hand Cases No. 55 and No. 56 agreed in the following points:—

(1) The loss of sensation followed lines incompatible with the areas affected in visceral disease.

(2) The plantar reflexes in Case No. 56 and the cremasteric and gluteal reflexes in Case No. 55 were present over the anæsthetic area.

(3) There was marked diminution in the visual field.

Now we can see how it is possible for the cerebro-spinal type to become associated with organic disease. The question at present agitating neurologists is the relation of disseminated sclerosis to "functional" disease. An enormous number of cases of disseminated sclerosis with tremor nystagmus and optic atrophy, have been preceded by years of undoubted "hysterical" manifestations—alteration in sensibility, vomiting, double sciatica, &c. Moreover, many of them have in this "hysterical" stage been "cured" more than once.

Thus I should like to suggest that the term "hysterical" be kept for the psychical type only, whilst the term "functional" disease be made to include all forms of generalised pain or loss of sensation following definite cerebro-spinal lines, whether the alteration in the nervous system is caused by anæmia, pyrexia, menstruation, &c., or some truly nervous influence, such as shock, or long continued visceral pain.

CHAPTER VI.

THEORETICAL.

§ 1. Why is pain referred?
§ 2. Why is the skin tender in visceral disease?
§ 3. The central relations of the various forms of sensation.

§ 1. *Why is pain referred?*

This constant reference of pain to the skin when the lesion affects an internal organ at first sight seems inexplicable. But I think a consideration of the following facts brings us a little nearer the comprehension of its significance and of the method in which it originally came about.

If I prick my foot I do not feel pain anywhere except in the foot itself about the point pricked. If the various parts of the body are tested it is found that our powers of localisation for a painful stimulus are very fairly accurate even over such comparatively insensitive parts as the back. If however the power of perceiving pain is diminished over a certain area on my foot, as occurs so frequently in such diseases as tabes dorsalis, the pain is no longer localised to the point pricked but is referred higher up the leg or if the sensation of the opposite foot is good to the corresponding point on the opposite foot. This is a phenomenon known as allocheiria. This is still better shown in cases of nervous disease where the patient has lost the sense of pain, to a considerable extent, over the areas of both sides of the abdomen corresponding to the distribution of the tenth and eleventh dorsal segments. If such a patient is pricked at a point about 2 inches below the umbilicus he will feel the pain but will refer it to a point about a similar distance

above the umbilicus. A second prick one inch below the umbilicus is referred to a point about the same distance above. A third prick almost on the border line which separates the area of diminished sensation from the area over which sensation is normal will be referred to a point just on the healthy side of the line. Thus, although the actual stimuli have been ascending towards the umbilical line, the patient says that he has experienced a series of pricks descending towards the umbilicus. If he is now pricked three times on the healthy skin over the spots to which he originally referred his pain, he will say that the pricks are ascending. Thus six painful stimuli applied over the abdomen, beginning at a point about 2 inches below the umbilicus in the area of diminished sensation and ending two inches above the unbilicus in the area of healthy sensation, will be said to be three pricks descending towards the umbilical line followed by three over the same spots ascending from the umbilicus.

This rule applies to all forms of sensation. If the sensation of touch is affected, but pain and heat and cold are normal, touch only will be referred, whilst heat, cold and pain are rightly localised. In the same way if heat or cold only are affected whilst pain and touch are normal, heat or cold stimuli will be referred, whilst pain and touch are rightly localised. I have records of cases in which only one form of the four sensations was wrongly localised, whilst the others were localised over the point of stimulation.

In all such cases of wrong localisation the particular sensation which is wrongly localised must be diminished, and must not be absolutely lost over the affected area. If the power of perceiving this stimulus is totally lost the stimulus is simply not perceived and there is no allocheiria.

Now the localisation of sensation is not a physical but a psychical phenomenon. It is not a question of the number of rods and cones or of the number of nerve end organs, but is a phenomenon of association. Thus supposing a prick was so applied that it just stimulated one nerve end organ, this stimulus so caused would pass up one fibre and probably remain distinct all up the cord. But in consciousness the

final result of this stimulus appears as a disturbance in the psychical field over a wide area with its maximum at one point representing the psychical unit which corresponded to the part stimulated. The disturbance over this point is, however, so greatly in excess of that over any portion of the diffusion field, that the diffusion field goes under the threshold and the position of the stimulus is rightly localised. If, however, the presentation to consciousness, which is the psychical equivalent of the point of stimulation, is less powerful than usual, owing to the physiological diminution produced by the partial analgesia of the area stimulated, the diffusion field will no longer be driven under the threshold. Thus the psychical effect produced by the area of diffusion may be even in excess of that representing the point of stimulation, and the latter will now go under the threshold. Under such conditions the position of the stimulation will no longer be localised at the point stimulated, but somewhere in the area of diffusion.

Now whenever an area representing one or more spinal segments on one side of the body is partially anæsthetic, whilst the sensation of the areas representing the same segments on the other side is normal, a stimulus to the skin over the affected areas is referred to the corresponding point on the normal side. If, however, the segments on both sides are affected, the stimulus is referred to the area corresponding to the area next above or next below the affected segments in their origin from the cord. Thus supposing the first, second and third lumbar areas are partly analgesic, a prick over their distribution will be referred to the area supplied by the twelfth dorsal or fourth and fifth lumbar. Now, as can be seen from plates I. and II., the posterior limits of these three areas closely abut on the limits of the second, third and fourth sacral roots on the outer and posterior aspects of the thigh. And yet a prick near the posterior border of these lumbar areas is not referred a few inches further back on to the distribution of the sacral areas, but to some point in the groin or below the knee (twelfth dorsal or fourth and fifth lumbar), more than 12 inches distant. This statement appears to be

true for pain, heat and cold, which do not overlap, but is I believe not true for touch, for the distribution of the touch areas overlaps considerably.

These facts have an obvious bearing on the reference of the pain produced by visceral disease. For the viscera are notoriously insensitive to touch and to the stimulus of a cut with a knife. Thus after gastrotomy the mucous membrane of the stomach can be tickled with a feather, or gently pinched with the forceps without the patient's knowledge. In liver operations it is customary to stitch the liver to the abdominal wall, as a preliminary to opening the organ when adhesions have formed. If the second stage of the operation is done without an anæsthetic the patient is quite unable to say when the knife is plunged into his liver, and after the whole is over and the drainage tube in place, will ask "when the operation is going to begin." The same statement applies to the second stage of an inguinal colotomy, in which the gut is opened by a clean cut of the knife. The only stimuli which seem to act on the viscera are tearing or rending in character, in fact pressure stimuli.

Thus, as our viscera are so notoriously insensitive, and as we have never had the opportunity of developing the sense of localisation in them, owing to their inaccesibility to touch, it is not to be wondered at that the maximum pain is not felt in the organ affected. They are thus as regards the localisation of sensation in exactly the position of the area on the skin whose sensation of pain was diminished in comparison with that of its neighbours. A painful stimulus to an internal organ is conducted to that segment of the cord from which its sensory nerves are given off. There it comes into close connection with the fibres for painful sensation from the surface of the body which also arose from the same segment. But the sensory and localising power of the surface of the body is enormously in excess of that of the viscera, and thus by what might be called a psychical error of judgment the diffusion area is accepted by consciousness, and the pain is referred on to the surface of the body instead of on to the organ actually affected. I do not mean to state that pain is never referred to the organ affected. Far from

it. The pain is frequently felt in the organ itself, but it is "dull," "heavy," "wearing," and not "sharp," "aching," "stabbing," like the referred pain.

Thus I believe that the phenomena of allocheiria and of referred pain in visceral disease are in nature and explanation essentially the same. Both depend for their appearance on the law that where a painful stimulus is applied to a part of low sensibility in close central connection with a part of much greater sensibility the pain produced is felt in the part of higher sensibility rather than in the part of lower sensibility to which the stimulus was actually applied.

§ 2. *Why is the Skin Tender in consequence of Visceral Disease?*

If impulses pass up the sensory nerves from an organ which is diseased to the cord they will set up a disturbance in the segment of the cord to which they are conducted. Now any second sensory impulse from another part conducted to the same segment of the cord will be profoundly altered. Under normal circumstances it would have set up its own proper disturbance in the grey matter and this disturbance would have been conducted to the brain. But now it no longer falls into a normal and quiescent cord but into one whose activity is already disturbed. The resultant stimulus conducted upwards is therefore a very different one from that which would have passed on under normal circumstances. In many cases the second stimulus will be exaggerated like rays passing to the eye through a convex glass. Thus if any segment of the cord is disturbed by painful stimuli from an internal organ a stimulus applied to the skin over the areas supplied by the nerve roots belonging to this segment will be exaggerated and a stimulus which normally was perhaps only uncomfortable would now appear to be very painful.

But in order that this condition may result the central connection between the sensory nerves from the viscera must be closely connected with that part of the cord into which the sensory stimuli from the skin are conducted. Now we never find cutaneous *hyperæsthesia* or increased sensitive-

ness to touch as the result of visceral pain but only increased sensitiveness to pain or *hyperalgesia*. It might be objected that we do not know of the possibility of hyperæsthesia apart from hyperalgesia but the following case illustrates the difference well.

A tabetic who is now under observation has complete loss of the sensation of pain over the thorax and abdomen. Thus he is quite unable to feel the prick of a sharp needle, and no reflex is produced by a sharp prick on the abdomen. But the slightest touch produces the most profound disturbance; an enormous reflex is produced and he says that the touch causes a feeling of the most exaggerated tickling. He complains that nothing he has ever experienced was equal to the exaggerated feeling of "ticklishness" which is produced by a light touch over the affected area. Thus the man has over the same area hyperæsthesia or exaggerated sensation of touch with analgesia or absent sensation to pain.

In the same way I have seen cases where even the mildest heat was said to "burn," though touch, pain and cold were normal; in such cases the reflex produced by the warmth was exaggerated whilst the reflexes produced by the other forms of stimulation were normal.

Now visceral pain always produces exaggeration of the sense of pain and sometimes exaggeration of the sense of heat and cold in the skin but never so far as my experience goes does it produce exaggeration of the sense of touch or hyperæsthesia proper.

§ 3. *The Central Relation of the Various Forms of Sensation.*

This leads me to another and wide-reaching question—What is the relation in the central nervous system of the various forms of sensation?

We have already seen reason to believe that the cutaneous distribution of touch overlaps considerably. On the other hand the cutaneous distribution of pain, heat and cold do not overlap appreciably. In this their distribution agrees with that of the trophic nerves to the skin from each segment

of the cord, which, as I showed by a study of the distribution of herpes zoster, are also found not to overlap.

Then again, we find that visceral disturbances may cause exaggeration of the cutaneous sensibility to pain, to heat and to cold, and are also closely related to trophic disturbances on the skin, such as herpes zoster. On the other hand they never cause exaggeration of the sensibility to touch.

Now in central gliomatosis, usually known under the name of syringomyelia, pain, heat and cold are profoundly disturbed, and there is a particular proneness to the development of cutaneous trophic lesions. On the other hand touch is either not affected at all or affected very late in the disease when the growth has produced profound pressure effects. Thus here again we find that a lesion around the central canal of the spinal cord tends to destroy the one group and to leave the other unaffected.

Thus I think that these facts tend to show that there is an intimate connection between the central connections for the sensory nerves of the viscera and the nerves which supply the sensation of pain, heat and cold, and also those which exert a trophic influence on the skin. I do not therefore think we shall be far wrong in assuming that the trophic nerves to the viscera bear a similar relation to the sensory sympathetic fibres as the trophic nerves to the skin bear to the fibres for pain. If this be so it will help to explain the effect produced by counter-irritation of the skin on the activity of the viscera. Conversely it will also explain the occasional improvement in pains of a purely nervous origin by some visceral irritation, *e.g.*, the removal of lightning pains over the 1st, 2nd, 3rd and 4th sacral [rectum] areas in a tabetic by the administration of a purge such as castor oil.

Thus to sum up I think we may conclude that the central connections of the pain fibres from the skin and viscera are closely connected with one another. The central connections of the nerves for heat and cold, and for trophic disturbances in the skin must also be in somewhat close association, though probably not actually connected. On the other hand

the nerves for touch from the skin (we do not know whether nerves for touch exist in the viscera) are widely separated centrally from those of pain. As to their paths in the cord I do not yet feel qualified to speak definitely.

It is now my pleasing duty to thank those who have been so liberal in their help during this investigation. Firstly, my best thanks are due to the physicians and surgeons of University College Hospital for the kind way they have permitted me to use their cases. I also have to return my sincere thanks to the medical and surgical staff of the National Hospital for Paralysed and Epileptic, Queen Square, and especially to Dr. Buzzard, my chief, for their generosity towards me. In each report from these two hospitals I have given the name of the physician and surgeon to whose kindness I am indebted for the case. In all cases the disturbance of sensation was worked out by myself, but in the surgical cases I am indebted to Mr. G. B. M. White, Surgical Registrar to University College Hospital, for the report of the patients' physical condition. I beg to return him my best thanks. My thanks are also due to Dr. Bowman and Dr. Wood, House Physicians at Queen Square, and to the House Physicians and Surgeons of University College Hospital for much kind thought and help in this laborious investigation.

PLATE I.

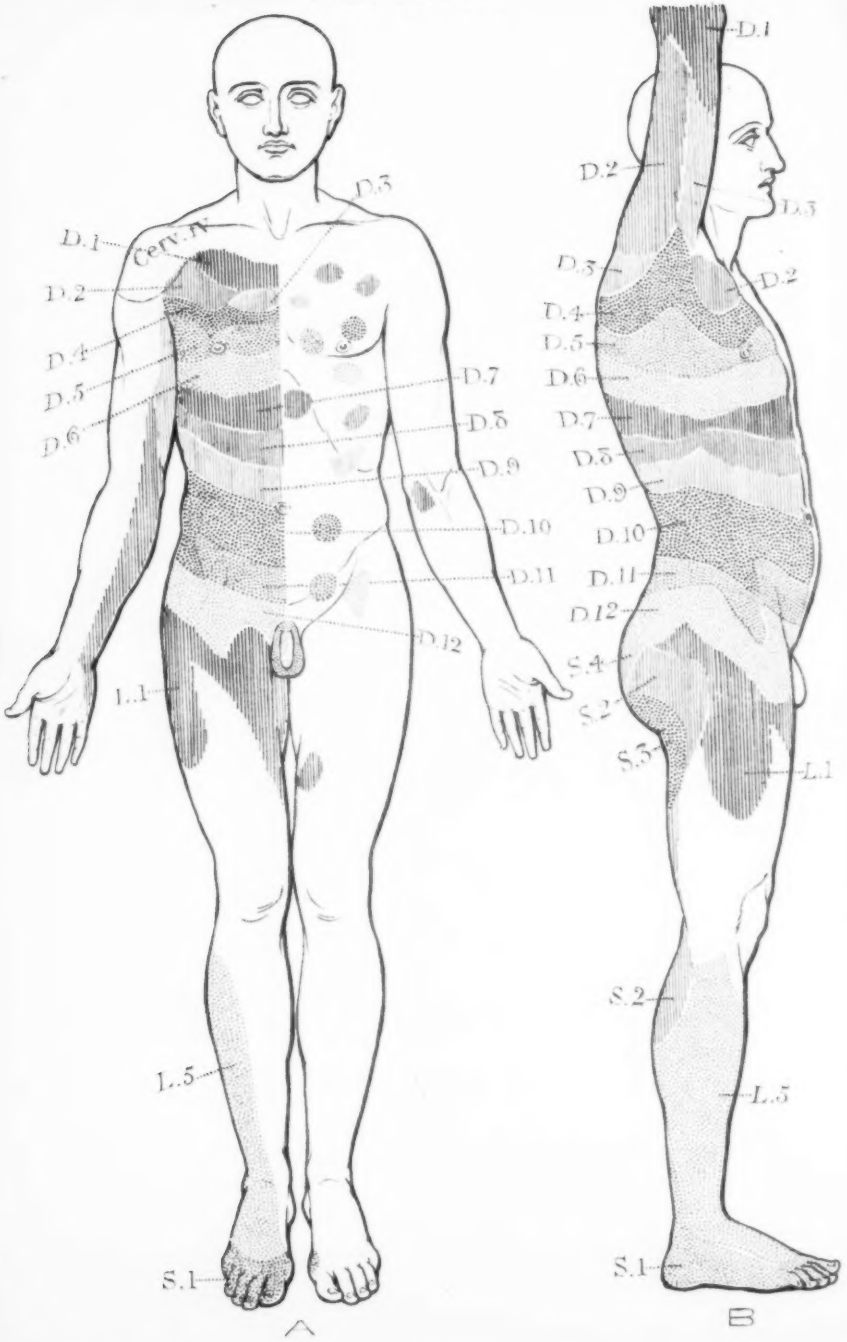
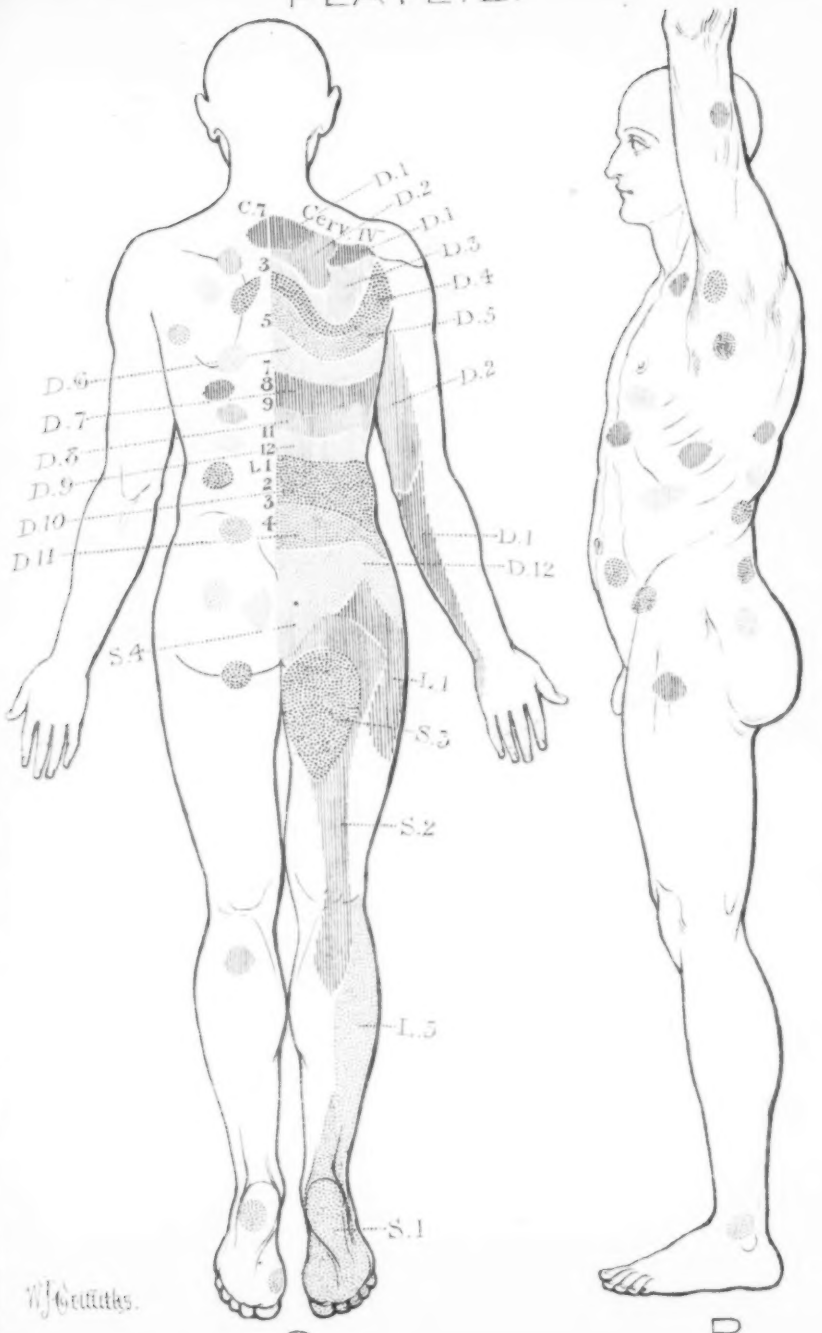

D.1
Cerv.N
D.2
D.3
D.4
D.5
D.6
D.7
D.8
D.9
D.10
D.11
D.12
L.1
L.5
S.1
A
D.1
D.2
D.3
D.2
D.4
D.5
D.6
D.7
D.8
D.9
D.10
D.11
D.12
S.4
S.3
L.1
S.2
L.5
S.1
B

PLATE. II.


C7
Cerv. IV
D.1
D.2
D.1
D.3
D.4
D.5
D.2
D.6
D.7
D.8
D.9
D.10
D.11
3
5
7
8
9
11
12
L.1
2
3
4
D.1
D.12
S.4
L.1
S.3
S.2
L.5
S.1
W. J. Griffiths.
C
D
Bale & Son, lith.

DESCRIPTION OF PLATES I. AND II.

These plates are intended to show the distribution of the segmental areas from the 1st dorsal to the 4th sacral. The form and extent of each area here figured is deduced from:

[1st]. The appearances presented by the cutaneous tenderness in visceral disease.

[2nd]. The distribution of the eruptions in 62 cases of herpes zoster.

[3rd]. The limits of analgesia (or loss of painful sensation) in organic disease of the spinal cord and roots.

I do not suppose that the areas here figured are absolutely correct, and can but hope that future investigation will show that their situation and extent bears a fairly close relation to the truth.

Cerv. IV. represents the lower border of the area supplied by the 3rd and 4th cervical areas.

Then follows a gap which is not usually represented in the disturbances of viscera. This gap corresponds to the 5th, 6th, 7th, and 8th cervical segments, and will be filled in, in my paper on the "Head and Neck."

D. 1 represents the dorso-ulnar or 1st dorsal area.
D. 2 represents the dorso-brachial or 2nd dorsal area.
D. 3 represents the scapulo-brachial or 3rd dorsal area.
D. 4 represents the dorso-axillary or 4th dorsal area.
D. 5 represents the scapulo-axillary or 5th dorsal area.
D. 6 represents the sub-scapulo inframammary or 6th dorsal area.
D. 7 represents the sub-scapulo ensiform or 7th dorsal area.
D. 8 represents the middle epigastric or 8th dorsal area.
D. 9 represents the supra-umbilical or 9th dorsal area.
D. 10 represents the sub-umbilical or 10th dorsal area.
D. 11 represents the sacro-iliac or 11th dorsal area.
D. 12 represents the sacro-femoral or 12th dorsal area.
L. 1 represents the gluteo-crural or 1st lumbar area.

Then follows a second gap which is not primarily represented in the disturbances of viscera. It corresponds to the 2nd, 3rd and 4th lumbar areas. A diagram showing this tract of skin split up into its segmental constituents will be found on page 53.

L. 5 represents the fibulo-dorsal or 5th lumbar area.
S. 1 represents the soleal or 1st sacral area.
S. 2 represents the sciatic or 2nd sacral area.
S. 3 represents the gluteo-pudendal or 3rd sacral area.
S. 4 represents the coccygeal or 4th sacral area.

PLATE I.

A. On the left-hand side of this figure the areas are shown from the front in their entirety. On the right-hand side the maximum spots of each area are figured, and in each case the colour is the same as that of the area to which each spot belongs.

B. This figure gives a lateral view of the areas marked in full on the left-hand side of fig. A.

PLATE II.

C. On the right-hand side of this figure the areas are shown from the back in their entirety. On the left-hand side the maximum spots of each area on the posterior aspect of the body are represented.

D. Shows a lateral view of the body on which the maximum spots of each area are marked as before.

To facilitate the recognition of these areas in any given case, I give a table on which are marked the viscera that are associated with tenderness in the various areas shown on these figures.

	HEART.	LUNG.	STOMACH.	INTESTINE.	RECTUM.	LIVER and GALL BLADDER.	KIDNEY and URETER.	BLADDER [Mucous Membrane and Neck].	BLADDER [Over distension and intermittent contractions].	PROSTATE.	EPIDIDYMIS.	TESTIS.	OVARY.	APPENDAGES.	UTERUS [In Contraction].	UTERUS [Lower Segment and Os Internum].
D 1	X	X														
D 2	X	X														
D 3	X	X														
D 4		X														
D 5		X														
D 6			X			X?										
D 7			X			X										
D 8			X			X										
D 9			X	X		X										
D 10				X		X	X			X		X		X		
D 11				X			X		X	X	X			X		
D 12				X			X		X	X?	X			X		
L 1							X		X		X			X		
L 5										X						
S 1								X?		X		·				X?
S 2					X			X		X					X	
S 3					X			X		X					X	
S 4					X			X							X	

THE NERVE CELL CONSIDERED AS THE BASIS OF NEUROLOGY.[1]

BY E. A. SCHÄFER, F.R.S.

IT is obvious that the nerve-cell must always form the basis of the science of Neurology in all its branches. But although this cell has been studied for a great number of years it is only now that we are beginning to arrive at a definite understanding regarding its structure, functions and relation to other nerve-cells. This has seemed to me therefore a suitable occasion for gathering up such threads of knowledge as we at present possess, and laying them before the members of this Society.

It is consequent upon the application of new anatomical and experimental methods that our knowledge of nerve-cells has lately become extended. Amongst anatomical methods which have yielded of late the most valuable results are the Wallerian method in its latest application by Marchi (23); the method of staining *intra vitam* by methyl blue which was introduced by Ehrlich (4); and the method of staining with nitrate of silver after treatment with chromic acid and its salts which was introduced by Golgi, and which has yielded invaluable results, not only in the domain of the nervous system, but in every other branch of histology. Amongst recent physiological methods the investigation by the galvanometer and electrometer of the paths taken by action-currents has in the hands of Gotch and Horsley (13) given results of great interest.

We may most naturally begin with the *structure* of the nerve-cell. I do not know why one should restrict the term "nerve-cell" to the body of the cell and thus exclude from that term the cell-processes. This is not done for any other kind of cell, and it appears to me

[1] Presidential Address delivered at the Annual Meeting of the Neurological Society, January 19th, 1893.

that the custom which has hitherto prevailed with regard to nerve-cells in this matter is not only inadvisable but even misleading. Waldeyer (45) has used the term "neuron" in this way to denote the whole nerve-cell, including all its processes. It is better, however, and simpler, to include under the term "cell," as is done with every cell of the body, the processes as well as the body of the cell. It will therefore be understood that by the term "nerve-cell" I shall denote not only, as has hitherto for the most part been the case, the body of the cell or the part immediately enclosing the nucleus, but all the several processes of the cell, and in that sense the word "nerve-cell" will be here used as identical with Waldeyer's term "neuron." The term "neuron" is short and convenient, but I shall employ it in a different sense, as will hereafter be seen, and I adopt this course the more readily because it has not yet come into very general use in Waldeyer's sense, and it is, I think, undesirable that it should do so.

The nucleus of the nerve-cell is noteworthy as compared with the nucleus of other cells by reason of its size, and it is large in proportion to the cell, *i.e.*, not only absolutely but relatively. If we examine a number of nerve-cells we find that the size of the nucleus and of the cell-body often bear a direct relation to the length of the processes which leave the cell. This is the case with the motor cells of the spinal cord and with the large projection-cells which are found in that part of the cortex of the brain from which the fibres of the pyramidal tract originate; it is also true for the ganglion-cells of the retina and the cells of the spinal ganglia. But I am bound to add that it does not appear to be an invariable rule, at least I doubt if it will apply to the visceral and vascular nerves. With regard to the structure of the nucleus the most important point in connection with this is the prominence of the nucleolus. This has long been noticed; in fact, one of the characteristics of a nerve-cell given in text books is that it contains a large and distinct nucleolus. There is also an intranuclear network as in most other cells which are not dividing, but this network is less distinct and more difficult to demonstrate than in many other cells. As

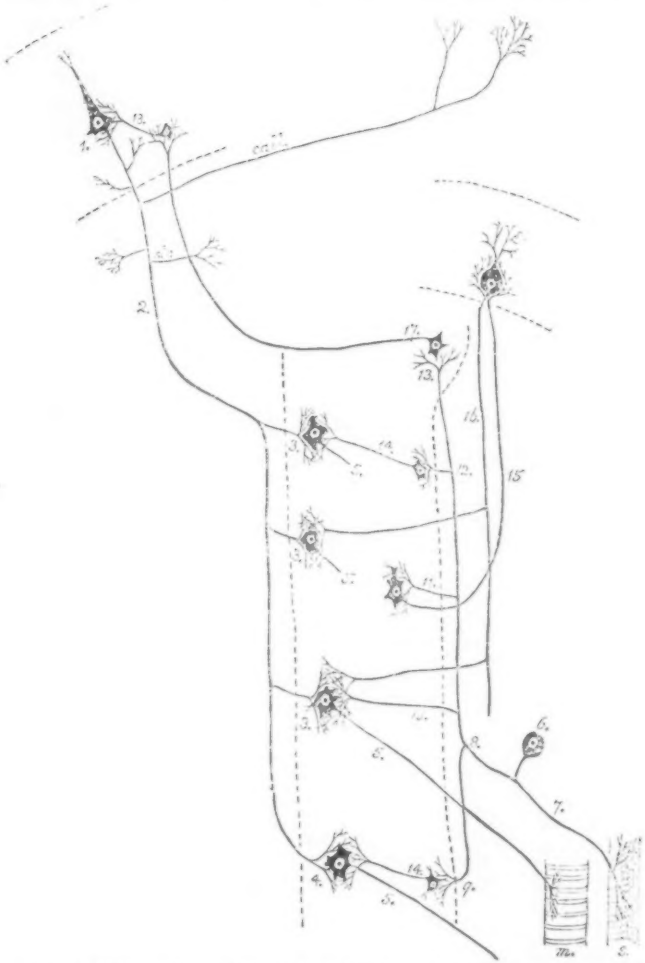
for the protoplasm of the cell-body there is not much that is new to remark. The usual reticular appearance has been described in this as in the protoplasm of other cells (21, 25); and it is also well known that the fibrils which can be traced in the processes of the cell can also be traced into and even through the protoplasm. We may note also a point which distinguishes these cells from many others, the frequency, namely, with which we find pigment accumulated in the body of the nerve-cell. This fact has sometimes been interpreted to imply decadence of function (8); but it seems to me that it is usually to be interpreted as a sign of activity rather than of decadence. For there is no doubt that in other organs the presence of pigment in cells is accompanied by marked protoplasmic activity, which may be both chemical and physical. I need only mention in this connection the hepatic cells, the pigment-cells in the skin of the frog and the hexagonal-cells which form the outermost layer of the retina of the eye. And it appears to me that the relative abundance of pigment in adult human nerve-cells as compared with nerve-cells of the child and of the lower animals is rather an indication of functional activity than the reverse.

We may next consider the processes of the cell. Every nerve-cell has one or more; it is an absolute characteristic of nerve-cells to possess processes. The processes are of two kinds. The first and the only essential kind is that which has long been known as the axis-cylinder or nerve-fibre process (Deiters). It is also the first kind to show itself in the course of development of the nerve-cell (14). The other kind is that which was distinguished by Deiters as the protoplasmic process. This is not so essential, for we find many nerve-cells entirely destitute of it. A word is here again necessary with regard to nomenclature. The above terms have been long in use to indicate these two kinds of processes, but they are obviously inconvenient from their length and from the assumptions which they appear to imply. I propose therefore to term the axis-cylinder or nerve-fibre processes *neurons*; and the protoplasmic processes *dendrons*.[1]

[1] The latter have been known as "dendrites," but the termination "on" is for various reasons preferable to "ite."

FIG. 1.

DIAGRAM SHOWING THE PROBABLE RELATIONS OF SOME OF THE PRINCIPAL CELLS AND FIBRES OF THE CEREBRO-SPINAL SYSTEM TO ONE ANOTHER.



1, a cell of the cortex cerebri; 2, its axis-cylinder or nerve-process (neuron) passing down in the pyramidal tract, and giving off collaterals, some of which, 3, 3, end in arborisations around cells of the anterior horn of the spinal cord, the main fibre having a similar ending at 4; *coll.*, a collateral passing in the

corpus callosum to the cortex of the opposite side; *str.*, a collateral passing into the corpus striatum; 5, axis-cylinder process (neuron) of anterior cornu-cell passing to form a terminal arborisation in the end-plate of a muscle-fibre, *m*.

6, a cell of one of the spinal ganglia. Its neuron bifurcates, and one branch, 7, passes to the periphery to end in an arborisation in the sensory surface, *s*. The other (central) branch bifurcates after entering the cord (at 8), and its divisions pass upwards and downwards (the latter for a short distance only); 9, ending of the descending branch in a terminal arborisation around a cell of the posterior horn, the neuron of which, again, ends in a similar arborisation around a cell of the anterior horn; 10, a collateral passing from the ascending division directly to envelop a cell of the anterior horn; 11, one passing to envelop a cell of Clarke's column; 12, a collateral having connections like those of 9; 13, ending of the ascending division of the posterior root-fibre around one of the cells of the posterior columns of the bulb; 14, 14, neurons of cells of the posterior horn passing to form an arborisation around the motor cells; 15, a fibre of the ascending cerebellar tract passing up to form an arborisation around a cell of the cerebellum; 16, neuron of this cell passing down the bulb and cord, and giving off collaterals to envelop the cells of the anterior horn; 17, neuron of one of the cells of the posterior column of the bulb passing to the cerebrum, and forming a terminal arborisation around one of the smaller cerebral cells; 18, neuron of this cell, forming an arborisation around the pyramidal-cell, 1. All the cells here represented, except 6, also possess dendrons, but these are mostly represented as cut short near the body of the cell, so as to avoid complicating the diagram.

And combined processes, such as occur in the motor nerve-cells of arthropods (31) can conveniently be known as *neurodendrons*. It must clearly be understood however that all processes of nerve-cells are ultimately dendritic. Almost without exception the neuron or nerve-fibre process, although it may have a course of several feet without giving off a branch, finally ends in a terminal arborisation (fig. 1). Examples of this fact occur readily: *e.g.*, the arborisation of the nerve-ending in muscles: the arborisation of nerve-fibrils in sensory structures, such as the cornea of the eye, and the epidermis of the skin. Even when a nerve-fibre apparently ends in a simple extremity it can generally be noticed, as in the corpuscles of Pacini, and in the tactile corpuscles of Meissner, that the actual ending of the essential part of the nerve-fibre is really arborescent.

It has been usual to classify nerve-cells according to the number of processes which pass off from the cell-body into uni-polar, bi-polar and multi-polar.[1] This classification is not,

[1] Cajal (30) has recently spoken of "apolar" nerve-cells, but this is completely a misnomer, for the cells in question (spongioblasts of the retina) have a well-marked process, which ramifies dendritically. What Cajal intends to imply is that they are devoid of an axis-cylinder process, but it appears to me that the nervous nature of these cells is very doubtful.

however, a natural one and does not distinguish between the kinds of processes given off from the cell-body, which ought to be done in any system of classification. Thus there are some nerve-cells which have no dendrons; there are others which have many dendrons. All, however, possess at least one neuron. We may then primarily distinguish between *dendric* and *adendric* cells; and according to the number of neurons we may further distinguish these cells into those which are *mononeuric* and those which are *polyneuric* (*dineuric*, *trineuric*, etc.), according as we find one, two, or more neurons or axis-cylinder processes passing off from the cell-body or from any of the dendrons. The majority of cells which have been hitherto observed belong to the mono-

FIG. 2.

A NERVE-CELL FROM THE SUPERFICIALOR MOLECULAR LAYER OF THE CEREBRAL CORTEX (after R. y Cajal.)

n, *n*, neurons; *dr*, *dr*, dendrons.

neuric class. As examples of this we may take the cells of the anterior horn of the spinal cord, many of the cells of the cerebral cortex, and the large cells of Purkinje of the cerebellum. But on the other hand, we have examples of cells with more than one axis-cylinder process (fig. 2) in some of the superficial cells of the cerebral cortex (29), and obviously also in the well-known "bi-polar" cells of the spinal ganglia of certain fish, and of the embryos of vertebrata generally, in the nerve-cells of the neuro-muscular sheet of Aurelia (43), and in other cells which I will not stop now to enumerate.

Starting, then, with the two kinds of nerve-cell, dendric and adendric, we can classify them further into those which are dendric and mononeuric, such as the cells of the anterior horn of the spinal cord, those which are dendric and poly-

neuric, such as some of the superficial cells of the cerebral cortex already mentioned; those which are adendric and mononeuric, as in the spinal ganglia of man, and those which are adendric and polyneuric such as the dineuric or bipolar cells above mentioned. The cells of sympathetic ganglia have hitherto usually been believed to be adendric and polyneuric, but we must now, as it would appear from recent observations of Cajal (30), Retzius (34), and Gehuchten (7), consider them as in reality dendric and mononeuric (fig. 3). In this way we obtain a classification of nerve-cells, firstly according to the kinds of processes which they possess, and secondly according to the number of neurons: the number of dendrons appears to be of altogether subordinate importance.

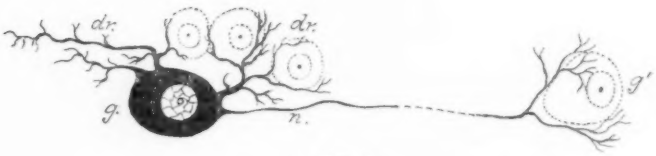


FIG. 3.

DIAGRAM OF A SYMPATHETIC GANGLION CELL (after G. Retzius.)

g., body of the cell; *dr.*, its dendrons; *n.*, its neuron, ending in a ramification which invests another cell (*g'*).

Nerve-cells may be further distinguished into those which possess relatively short neurons, and those which possess relatively long neurons. This distinction gives rise to the two types of nerve-cell which were first described by Golgi (9, 10, 11), and which were by him regarded as motor and sensory respectively. As is now well known, Golgi regarded the cells which possess long axis-cylinder processes (*e.g.*, the cells of the anterior horn of the spinal cord, and the projection-cells of the grey matter of the cerebral cortex) as being concerned mainly in the giving out of efferent impressions; whereas he regarded the cells (usually smaller in size) possessing relatively short and soon-branching nerve-fibre processes, as essentially sensory or receptive in function. But there can be little doubt that this distinction into motor

and sensory cells which was drawn by Golgi, can no longer be accepted. At the same time there may still be a functional difference between the two kinds of cell, but this functional difference may perhaps better be expressed by using the term *projection-cell* for the cell of Golgi's first type with long axis-cylinder process, and the term *intermediary cell* for the cell belonging to Golgi's second type, with relatively short axis-

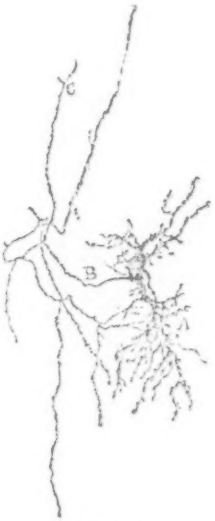


FIG. 4.

AN INTERMEDIARY NERVE-CELL FROM THE POSTERIOR HORN OF THE SPINAL CORD. (R. y Cajal).

A, dendrons; B, neuron, soon ramifying, and its branches ending either simply or in bifurcations (as at *c*).

cylinder process. By the latter term is implied that the cell in question offers an intermediary link between centripetal impressions which may be brought to a nerve-centre by the neuron of a sensory projection-cell, and the centrifugal impressions which pass away from the nerve-centre by the neurons of motor projection-cells.

With respect to the structure of the cell-processes, it is impossible to say positively that there is any essential

difference between the neurons and dendrons, but there are certain minor differences which can be noted. One of these is the mode in which lateral processes are given off from main trunks. From the dendrons these usually come off at triangular enlargements, from the neurons without such enlargements. The dendrons usually begin to branch quite close to the cell-body, but the neuron also, which was supposed at one time not to branch until it reaches its termination, often gives off fine lateral ramuscles very early in its course. To such fine branches of the neuron, wherever occurring, the name of *collaterals* has been given by Cajal; this is a convenient term and one which may very well be adopted. There is further often some difference in appearance between the two kinds of process when prepared by the Golgi method of staining, so that an experienced observer can detect in the midst of an interlacement of fine fibres derived from the ramifications of the dendrons of nerve-cells, neurons of the same or of other cells, traversing the grey matter through the interlacement; but it is not easy always to say what it is which confers this difference of appearance. It is, perhaps, due to the fact that the neurons remain almost unaltered in diameter along their course, whereas the dendrons, from the manner in which they branch, become gradually smaller as they are traced inwards. Moreover, the dendrons have usually a rough outline, while the neurons are more commonly smooth. Some of these differences are very manifest in the cells of the cerebral cortex (fig. 5).

Both neurons and dendrons have an apparently fibrillar structure. This structure has been mainly investigated in the neurons as they occur as axis-cylinders of the medullated nerve-fibres. The current idea of the structure of the axis-cylinder of the nerve-fibre, is that it is composed of a bundle of fine fibrils, which form the true conducting part of the nerve-fibre, and which are embedded in a clear substance comparable to the clear substance of protoplasm. These fibrils—ultimate fibrillæ of Max Schultze—have usually been regarded as completely distinct from one another; but the fact must not be ignored, that in the opinion of many

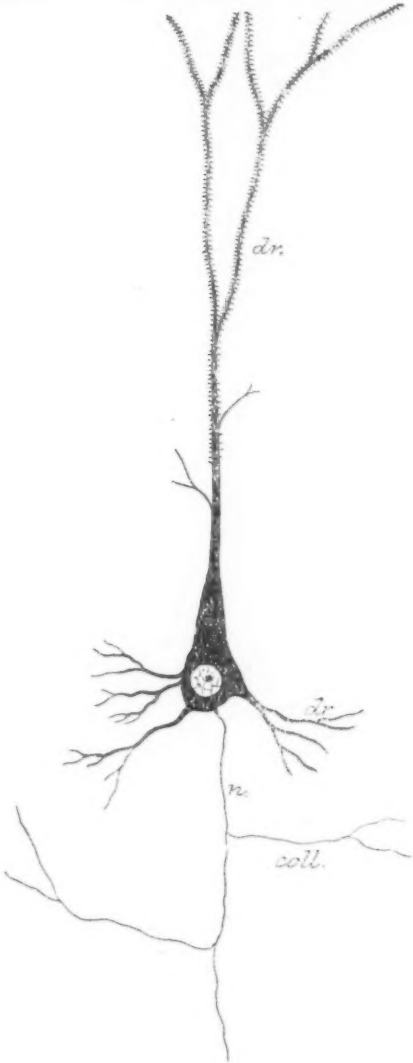


FIG. 5.

A PROJECTION-CELL OF THE CEREBRAL CORTEX.

dr, dendrons; *n*, neuron; *coll.* collaterals.

histologists (Heitzmann, Leydig, Frommann, Nansen) the nerve-fibrillæ of the axis-cylinder are nothing but a repetition of the reticulum of fibrils which is described as occurring within the protoplasm of all cells; the reticulum having, in this case, been drawn out to such an extent that the meshes of the reticulum become extremely elongated and the fibrils

FIG. 6.

VARICOSE NERVE FIBRILS SEEN WITHIN AN AXIS-CYLINDER. (M. Schultze).

of the reticulum, to all appearance, parallel and distinct. In fact, according to Nansen (25), the apparent fibrils are really the optical longitudinal sections of sheaths or septa of spongioplasm, which subdivide the fibre into tubes filled with

FIG. 7.

VARICOSE NERVE-FIBRILS AS SEEN IN A CHLORIDE OF GOLD PREPARATION OF THE CORNEA. (Ranvier).

hyaloplasm, which forms the true conducting material of the nerve-fibre. The observations of Max Schultze, however, upon the structure of the non-medullated fibres of the olfactory nerve and of the axis-cylinder of the ordinary medullated fibres (fig. 6), indicate, with considerable clear-

ness, an anatomical discontinuity of the individual fibrils, and this is further borne out when we trace the ramification of the nerve-fibre at its peripheral extremity. Here, in many instances, as for example in the cornea (fig. 7), and even in the nerve-endings of the motor-nerves upon the so-called end-plates, we can observe a complete separation of the fibrils which have composed the axis-cylinder and which are here ending in the ultimate branchings of the axis-cylinder.

I have here a representation of a photograph from a section of nerve-fibres in which the fibrils appear distinct from one another, and tubular in structure. In the photograph, which was taken with an apochromatic

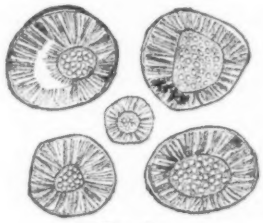


FIG. 8.

SECTION OF NERVE-FIBRES SHOWING THE TUBULAR APPEARANCE OF THE FIBRILS OF THE AXIS-CYLINDERS.

oil-immersion objective of Zeiss, the section of each fibril shows a minute circular clear area bounded by a definite line. I know no other way of interpreting this appearance than by assuming it to be really a minute tubule. And it seems to me also that the appearance of the terminating fibrils, as we see them in nerve-arborisations which have been treated by various re-agents, and as they have been represented by Max Schultze in broken axis-cylinders after treatment with osmic acid, bears out this conclusion, for it is difficult otherwise to understand why we should invariably find upon such nerve-fibrils the varicosities which every histologist knows to be so characteristic of them. It is easy on the other hand to comprehend the production of such varicosities if we imagine the fibril to be an excessively fine-walled tubule

filled with fluid; in fact it would be almost impossible to suppose that under the influence of re-agents such minute and thin-walled tubules should not take on a varicose form. This is a question which we cannot at the moment decide; but it is one of some interest with regard to the possible manner in which nerve-impulses are conducted along nerve-fibres.

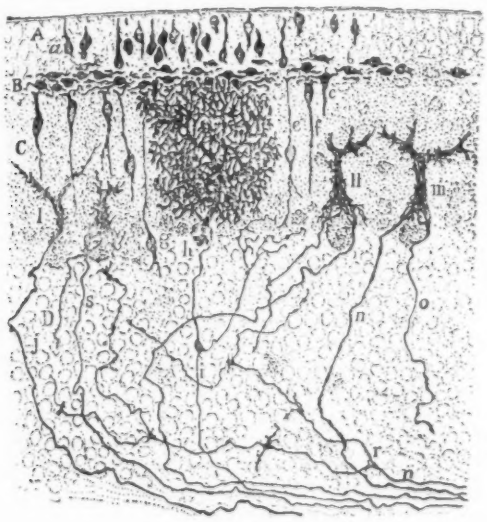


FIG. 9.

TRANSVERSE SECTION OF THE CEREBELLUM OF A 16-DAY OLD PUPPY, GOLGI'S METHOD. (Ramón y Cajal).

A, epithelium-like layer of superficial zone; B, layer of horizontal bipolar cells; C, molecular layer; D, granule layer.

a, epithelium-like cell; *b*, bipolar horizontal cell; *c*, cell sending a process downwards into the molecular layer; *e*, *f*, *g*, bipolar vertical cells; *h*, cell of Purkinje; *i*, its nerve-fibre process giving off a collateral towards the molecular layer; *j*, *n*, *o*, *s*, fibres from the white substance passing to form plexuses which envelop the upper part of the bodies and the proximal part of the dendrons of cells of Purkinje; *r*, "moss" fibre; *p*, *q*, moss-like expansions on its course and at its extremity.

The next part of our subject to come under consideration is the relation which nerve-cells bear to one another. It was formerly supposed that all nerve-cells were united with one another by distinct processes, and when it was shown by the

careful preparations of Gerlach and other observers, that this direct union of one nerve-cell with another by relatively coarse fibres or processes does not obtain, it was still believed that they became united by means of a fine network which was supposed to pervade the whole grey matter of the nervous system, and which was believed to be formed by the ramifications of the dendrons uniting with one another. We can now, however, state by virtue of observations which have accumulated since the employment of

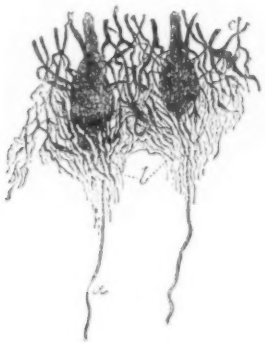


FIG. 10.

BASKET-WORK OF FIBRES AROUND TWO CELLS OF PURKINJE. (Ramon y Cajal.)

a, axis-cylinder or nerve-fibre process of one of the corpuscles of Purkinje; *b*, fibres prolonged over the beginning of the axis-cylinder process; *c*, branches of the nerve-fibre processes of cells of the molecular layer, felted together around the bodies of the corpuscles of Punkinje.

Golgi's method of staining, that firstly, there is never a direct union of nerve-cells by comparatively coarse fibres;[1] secondly, there is not even a union of nerve-cells by means of a net-work of fine fibrils; and thirdly, that every nerve-cell with all its processes is a distinct and isolated anatomical unit.[2] We can further state with great probability that the only connection of one nerve-cell with another

[1] The case of twin-cells, such as are found in the rod and cone layer of some animals, and such as Fritsch (5) has described in the spinal cord of the electric fish, Mormyrus, are only apparent exceptions to this rule.

[2] It was to this distinct isolated anatomical unit that Waldeyer proposed to devote the term "neuron."

is a physiological one, and that it takes place by the adjunction of the arborised process or processes of one nerve-cell, either with the cell-body of another cell, as in the cerebellar cortex (figs. 9, 10) and in various other parts, or by the adjunction and interlacement of the arborised processes of one nerve-cell with similar arborised processes of other cells, as in the

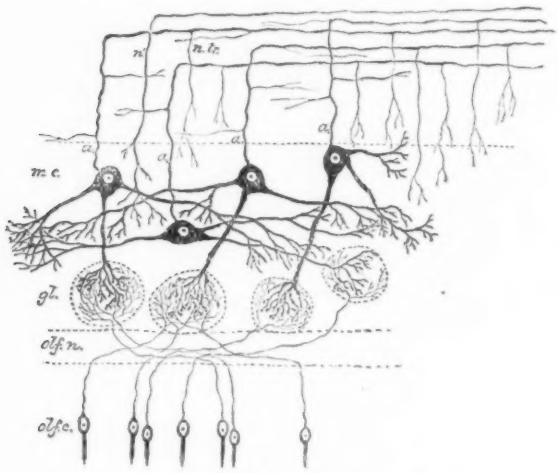


FIG. 11.

DIAGRAM OF THE CONNECTIONS BETWEEN THE OLFACTORY SENSE ORGAN AND THE BRAIN.

olf.c., olfactory cells sending their neurons *olf.n.* into the olfactory glomeruli, *gl*; *m.c.*, "mitral" cells of olfactory bulb, sending dendrons into the glomeruli, and neurons, *a*, *a*, into the olfactory tract, *n. tr.*; *n.'*, a neuron derived from a cerebral cell, ending in the olfactory bulb.

olfactory glomeruli (fig. 11, *gl.*) In fact we may regard the basis of the grey matter of the nervous system—the granular-looking substance in which the nerve-cells are embedded—as an extremely fine interlacement of ramified processes, not only of the nerve-cells which actually lie in that particular grey matter, but also of nerve-cells which lie in other parts of the nerve-centres or even in the peripheral parts of the nervous system, and which on arriving at the grey matter similarly break up into a fine arborescence of nerve-fibrils.

This view is extremely probable by reason of the numerous observations which have recently accumulated on the disposition of nerve-fibres and nerve-cells within the grey

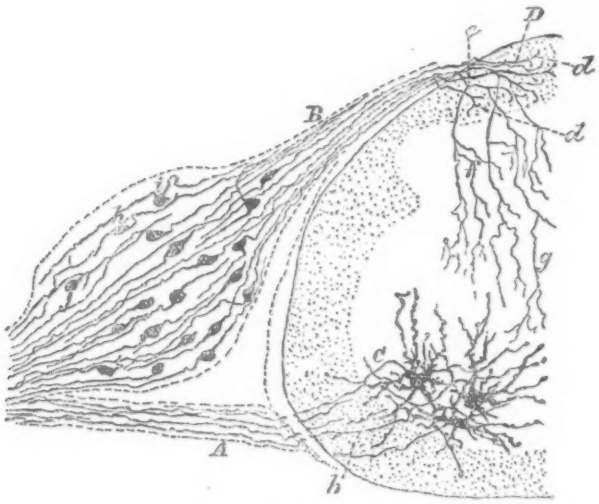


FIG. 12.

TRANSVERSE SECTION OF THE SPINAL CORD OF A CHICK ON THE 9TH DAY OF INCUBATION, PREPARED BY GOLGI'S METHOD. (Ramón y Cajal).

A, axis-cylinders of anterior root-fibres issuing from large cells of the anterior horn, *C*.

B, Posterior root-fibres passing from the bi-polar cells of the spinal ganglion, into the posterior column of the spinal cord, *D*, where they bifurcate, *d*, and become longitudinal; *e*, *f*, *g*, collaterals from these fibres, passing into the grey matter.

matter of the vertebrate nerve-centres (6, 17, 20, 27), and it is rendered still more probable by what we now know regarding the granular-looking substance which forms a large portion of the ganglia of the central nervous system of invertebrates and which was termed by Leydig, who first carefully described, it the *Punktsubstanz*. Within this "Punktsubstanz" (dotted substance) it has long been known that many of the processes of the nerve-cells terminate, and it is now recognised by the employment of new methods, and particularly the method of Ehrlich, and the method of Golgi, that the

Punktsubstanz is entirely made up of the finely ramified and somewhat varicose terminations, not only of the processes of the nerve-cells of the ganglia which constitute the central nervous system in these animals, but also of processes which arrive at those ganglia from nerve-cells at the periphery. I would refer you, in substantiation of this fact, to

FIG. 13.

DOTTED SUBSTANCE OF LEYDIG IN ASTACUS. (After a figure by G. Retzius).

the accompanying figure (fig. 13), after Retzius (31); showing the dotted substance in one of the ganglia of Astacus. In this it will be seen that the so-called Punktsubstanz is mainly made up of ramifications of fibres derived from the neuro-dendrons of the large motor nerve-cells. Similar observations have been made by Rohde (40) and by Biedermann (2).

We may next consider the *functions* of the nerve-cell, and in doing this it will be convenient provisionally to consider the functions of the cell-body and cell-processes distinctly.

The first and most prominent function of the body of the nerve-cell is unquestionably that of nutrition; and this function is in all probability dependent upon the presence of the nucleus. Many instances could be given from cells other than nerve-cells of the dependence of the nutrition of cell-protoplasm on the nucleus. Thus it has been shown

by Hofer that when an amœba is separated into two parts, one of which contains the nucleus and the other is deprived of a nucleus, the part which contains the nucleus will continue to live, to take in nourishment, to grow; will in fact continue to perform all the functions of the cell; whereas the part which is deprived of the nucleus, although it does not immediately die, and although it may still exhibit in a less active manner many of the phenomena of amœboid movement, yet fails to take in nourishment, fails to form more protoplasm or another nucleus, and although it may continue for a time to exist, ultimately perishes, probably from inanition. It has also been shown that vegetable cells (or portions of such cells) which are deprived of a nucleus, although they also can continue to live for a time, no longer carry on their nutritive functions as before, and that such portions of cells, although they may comprise the greater part of the original cell, have entirely lost the power of forming a cellulose wall, a function which was possessed by the original cell, and is still maintained by the portion of cell which yet contains a nucleus. And it has lately been shown by Hodge (15), who subjected the fibres of the posterior roots of the spinal nerves to prolonged stimulation, that this continued activity of the fibres is accompanied by marked wasting of the nuclei of the cells of the spinal ganglion, as well as by certain alterations in the cell-protoplasm; changes which are probably produced by the increased activity of their trophic functions, for as we shall presently see, the nerve-impulses do not, in all probability, actually traverse the cell-bodies of these cells. It is not therefore surprising to find that when a portion of a nerve-cell, *e.g.*, one of its processes, is separated from the cell-body containing the nucleus, that portion of the nerve-cell, although it may continue for a time to live, and even to conduct nerve-impulses when these are artificially produced in it, ultimately dies. This is in fact the basis of what is known as the Wallerian degeneration of nerve fibres. When we completely recognise that a nerve-fibre is nothing but the process of a nerve-cell, we understand immediately how it is that when a nerve-fibre is severed the part which is cut off from

connection with the parent cell must ultimately die. This is found to be an invariable rule and it matters not whether the part of the fibre which is thus severed from the cell conducts impulses towards or from a nerve-centre; in both cases the result is the same—the process of the cell dies. It involves in its death, or at least in the changes which accompany its death, certain changes in the sheaths of the nerve-fibres which enable us with great readiness to recognise such degenerating fibres; and this the more easily from the method which Marchi has introduced, and by means of which we can stain deeply the myeline sheaths of such nerve-fibres in the earlier stages of their degeneration, whilst leaving unstained the myeline sheaths of normal fibres. This nutritive function of the body of the nerve-cell has not always been so completely recognised as it deserves, but has been of late strongly insisted upon by Nansen (25), who has even gone so far as to deny to the cell-body any other function than that of presiding over the nutriment of the whole cell and especially over that of the axis-cylinder process. It is clear, however, that although the function of nutrition may be the one essential function of the nucleated body of the nerve-cell, many cases occur in which the body of the cell also serves for the transmission of nerve-impulses. That must be so with the so-called bi-polar cells, where the cell-body gives origin to two nerve-fibres passing in opposite directions; the impulses which course along these nerve-fibres necessarily traverse the body of the cell. And this must also be the case in the motor projection-cells of the spinal cord and bulb where each nerve-cell gives off only one unbranched neuron. In this instance nerve-impulses are probably communicated to the body of the cell from the interlacement of nerve-fibres, derived from other cells, which enwrap the body of the motor cell. Granting, however, the possibility of the transmission of nerve-impulses through the cell-body it is quite open for us to believe that this is, if one may say so, more or less accidental. It is certain that there are many instances in which nerve-impulses can be conceived to be transmitted along nerve-fibres without in the course of that transmission passing through a nerve-cell at all. This is in all probability

the case with the cells of the spinal ganglia of higher vertebrates. From these cells, as is well known, only one process passes off and this process, as was first shown by Ranvier, bifurcates, one branch passing towards the periphery and the other towards the nerve-centre. Sensory impulses which originate in the periphery and pass along the fibre can perfectly be conceived as being conducted directly to the nerve-centre, past the T-shaped junction with the process of the nerve-cell, and without passing through the nerve-cell at all. It has indeed been actually shown by Langley and Anderson (18) that the passage of nerve-impulses along the fibres of the posterior roots is not blocked by a dose of nicotine, which would be more than sufficient to block their passage through sympathetic ganglia. And in the case of invertebrates, such as the earth-worm, the structure of whose nervous system has recently been elucidated by Lenhossék (19) and Retzius (32), it is clear that sensory impulses which are brought by the fibres from the integument to the central nervous system may perfectly well become converted into motor impulses within the Punktsubstanz without necessarily traversing the motor nerve-cells at all; the latter being only connected with the Punktsubstanz by dendritic collaterals, which pass off from their large neuro-dendric processes (fig. 14).

It is, however, almost universally held that nerve-cells may act as generators of nerve-impulses: that in fact they may function as independent nerve-centres. But it is by no means easy to bring forward direct proof of this action. Perhaps the so-called automatic action of nerve-centres would be regarded as the best instance of such intracellular genesis of nerve-impulses, and, as an example of automatic action, the action of the respiratory centre in the medulla oblongata (which continues to convey nerve-impulses along the efferent channels when all afferent channels to the cells have been cut) may be taken. But this action of the respiratory centre is probably due to the chemical stimulation which the centre receives from the venosity of the blood circulating in the vessels of the medulla oblongata. The stimulus is supposed to affect the nerve-cells which give

origin to the fibres passing towards the respiratory muscles, or at least to those spinal segments from which the fibres to the respiratory muscles arise: it practically amounts to an external stimulus applied to the nerve-cells and is

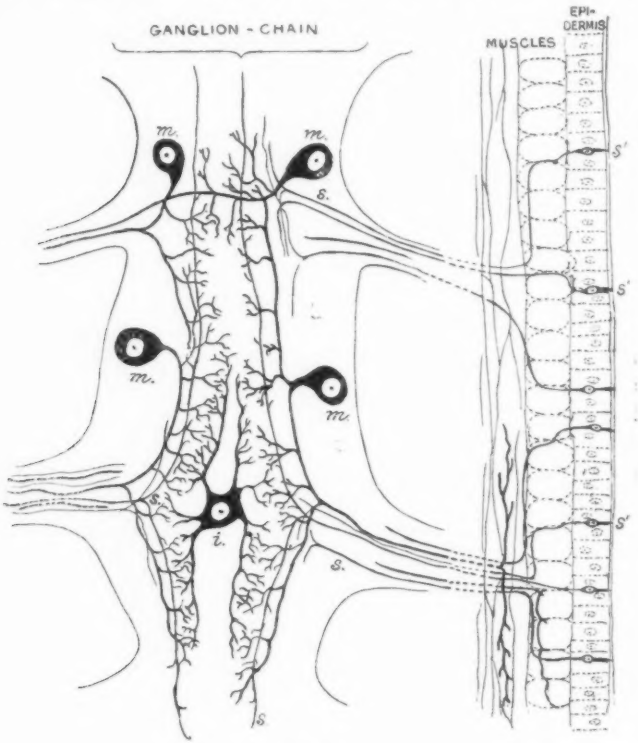


FIG. 14.

DIAGRAM OF THE NERVOUS SYSTEM OF LUMBRICUS. (After Lenhossék and Retzius).

m, motor nerve-cell; *i*, intermediary cell; *s*, sensory fibres proceeding from *s'*, sensory cells in the epidermis.

comparable to the application of an artificial stimulus to the same part of the nervous system, such, for example, as the chemical stimulus of salt, or the electrical stimulus derived from an induction coil. The action of the nerve-

cells cannot therefore be said to be truly automatic: in other words, the nervous impulses do not arise *de novo* without any obvious external cause by the action of the cell-substance. And I think that it would be difficult to instance any "automatic" action which might not also be referred either to a chemical stimulus due to variations in the blood or to stimuli reaching the centres from the periphery, or from some other parts of the nerve-centres.

There is no doubt, however, that on due stimulation, whether mechanical, chemical, or electrical, the body of a nerve-cell may produce nerve-impulses. This, of course, is only to be expected, since the processes of the cells are capable of producing nerve-impulses under similar conditions. It is a function which is not special either to the cell-body, or to the processes, but is common to both. We may, however, fairly consider whether there is no difference in the performance of this function in the cell-body as compared with its performance by cell-processes.

Unfortunately, our knowledge of nerve-impulses is extremely limited. We know their rate of transmission; that they are transmitted in a wave-like manner, and can pass in either direction along nerve-fibres; and, further, it is probable, although not absolutely proved, that the passage of nerve-impulses is accompanied by chemical processes. With regard to nerve-cells we have evidence of a more or less positive character on this head. It has been shown that the grey matter of the nerve-centres becomes acid after it has been excited by drugs or by other methods, and that it also, when it has been called into activity, produces a notable rise of temperature. This rise of temperature was shown to the Royal Society last year, by Mosso (24). At the same time, the opinion has been largely held that nerve-fibres themselves do not participate in these chemical processes of the nerve-cell. Points in favour of this view are chiefly derived from the fact that various observers, who have investigated the conditions of nerves during the passage of nervous impulses, have come to the conclusion that nerve-fibres cannot be fatigued.[1] And it is now believed that fatigue, both in

[1] The literature of this subject is referred to by Edes (3).

muscles and in the nerve-centres, is produced by the accumulation of the products of chemical processes; if, therefore, nerve-fibres show, even after prolonged activity during many hours, no sign of fatigue, it is probable that the chemical processes which produce fatigue within the nerve-centres, do not occur within the nerve-fibres. At the same time the latest observations upon this subject, those of Edes (3), have shown that after some hours of excitation the action-currents which result from the excitation tend to become weakened, and since these action-currents may be taken as a sign of nerve-activity, this would be an indication that even in peripheral nerves prolonged excitation may ultimately produce fatigue. Rolleston (42) endeavoured to obtain evidence regarding the same question in a different way. Determining by a delicate method whether the passage of nerve-impulses along the fibres is accompanied by a development of heat, he was unable to obtain evidence of any such warming. But it must be noted that Rolleston found that when the nerve was dying there was evidence of the formation of heat, and since the chemical processes which accompany death of living tissue appear to be very similar to the chemical processes which accompany activity, as is seen, for example, in the case of muscle, it is very possible that there may still have been thermal changes proceeding in the living nerve when excited, but that these were of too slight a nature to be detected owing perhaps to the very small amount of true conducting matter within a peripheral nerve as compared with the bulk of nerve-sheaths and connective tissue structures. Perhaps also for the same reason, as well as the fact that alkaline lymph surrounds the nerve-fibres, it is impossible to determine that a nerve which has been tetanized for some time shows any sign of acidity. But, as we have already noted, when the central nervous system is thrown into activity, the white matter, which is formed wholly of nerve-fibres, becomes acid as well as the grey cortex. Since a nerve-fibre is only a prolonged part of a nerve-cell, and since we know, firstly, that nerve-cells may undergo fatigue, secondly, that chemical processes go on both in the white and grey matter of nerve-centres; thirdly, that heat is

developed in grey matter, and that this heat is due, probably, to chemical processes, it is reasonable to suppose that similar chemical processes may go on in axis-cylinders of nerve-fibres but that, for the reasons we have above indicated, it has hitherto proved impossible to detect them. Moreover, the apparent non-fatigueability of nerve-fibres may be partly due to the ready reparative processes of the axis-cylinder, which may derive material for such reparation from the substance of the myeline sheath.

Before considering further the manner in which nervous impulses are produced within the bodies or the processes of nerve-cells, it may be well to enquire what is the nature of a nervous impulse. It is usually assumed that nervous impulses are either waves of molecular vibration or of chemical action (that is to say, of explosive change), or of the two combined. There is absolutely no evidence in favour of the first supposition, although, as we have just seen, there is some indication that they are accompanied by chemical action. We must not, however, ignore the possibility that they may be waves of pressure or surface-tension change. The supposition that the electrical phenomena of active muscle and nerve are produced by variations of surface-tension passing in a wave-like manner along the fibres, has, in fact, been recently insisted upon as probable by d'Arsonval (1). Such variations must necessarily occur when in an elastic tube with fluid or semi-fluid contents, pressure-waves are in any way set up. But we have already had evidence that the nerve-fibrils may be extremely fine tubules (compare p. 145), and in such tubules we can readily understand how waves of surface-tension change may be possible.

Assuming that nerve-impulses may be waves of pressure or surface-tension change, we may next conjecture in what manner they might originate. Such waves might be produced by the contraction of the nerve-cell, or of any of its processes. It is certainly true that except in the cone-cells of the retina, this contraction has not hitherto been observed; but, on the other hand, it may generally be so slight as to escape observation, or it may be confined to the fibrillæ. If such contraction were assumed to be rhythmic, we could

then understand how it happens that from many nerve-cells, nerve-impulses pass along nerve-fibres with a regular rhythm. This is seen in the motor nerve-cells of the spinal cord, for it is found that a rapid stimulation of the excitatory area of the brain or of the fibres of the pyramidal tract—a stimulation which applied to a motor nerve directly is sufficient to produce complete fusion of the muscular contractions, and therefore complete tetanus—is, when applied to the brain, or to the pyramidal tract, responded to by an incomplete tetanus of the voluntary muscles—a tetanus having a fairly regular rhythm of about 10 per second.

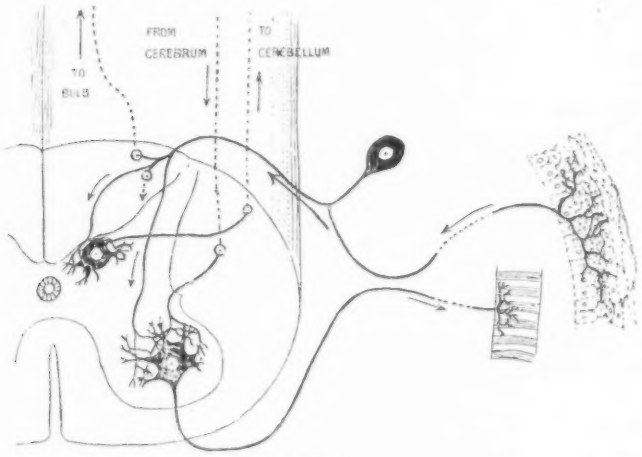


FIG. 15.

DIAGRAM OF THE RELATIONS OF MOTOR AND SENSORY CELLS WITHIN THE SPINAL SEGMENT, AND THEIR CONNECTIONS WITH THE HIGHER NERVE-CENTRES.

The annexed diagram (fig. 15) shows the anatomical mechanism which probably takes part in this experiment. When a fibre of the pyramidal tract is excited, the nerve-impulses which are generated in that fibre, and which are probably of the same rate as the excitation, pass down to the grey matter of the spinal cord, and are there converted into nerve-impulses which may have a very much less frequent rhythm. This can only, so far as appears, take place at the

adjunction of the terminations of the pyramidal-tract fibres with the motor nerve-cells; and it would appear that the motor nerve-cell is stimulated by the nerve-impulses which are conveyed along the fibre of the pyramidal tract, but that it responds to that action with a very much slower rhythm than that of the assumed excitation; for the excitation may be as rapid as 100 per second, or more, but provided it is not too intense the impulses which pass along the motor fibres are only at the rate, as shown by the response of the muscle, of about 10 per second. The same thing is seen when the muscles are made to contract by a reflex excitation of the skin. Such an excitation may be very rapid or it may even be continuous. This rapid or continuous excitation of the skin produces in the sensory fibre nerve-impulses which may be assumed to be at least as rapid as the excitation itself, and these are conveyed to the grey matter of the lower nerve-centres, and are converted into nerve-impulses of a relatively slow rhythm, as shown by the rhythm of the reflex muscular response. This transformation may be assumed to occur either in the motor projection-cell, or in an intermediary cell, if any such intervene between the afferent fibre and the motor-cell, and the slow rhythm of the epileptiform convulsions which follow strong electrical excitation of the cerebral cortex, and which certainly originate in the cells of the cortex, furnishes another well-marked instance of rhythmic production of nerve-impulses by nerve-cells.

There is another function of nerve-cells which we have yet to consider, and that is the reception of impressions from the exterior. All nerve-cells which receive impressions from the exterior, may provisionally be termed sensory cells although it does not necessarily follow that the impression may ultimately affect the sensorium. The sensory cells with which we are most familiar, are those of the spinal ganglia. Here (fig. 16, TACTILE) the sensory impression is not received by the body of the cell, but by a peripheral process, which passing either to a special end-organ such as a tactile corpuscle or end-bulb, or insinuating itself between the elements of the integumental and other structures, receives the impressions which cause nerve-impulses and transmits those

impulses upwards towards the nerve-centres. It is not only the case with those sensations which are received through the surface of the skin or by the action of the muscles, that sensory impressions are in the first instance communicated to processes of nerve-cells; for we find from the recent observations of Retzius [33, 37] that the same is true for the auditory and for the gustatory organ (fig. 16), the nerve-terminations in which do not, as was at one time

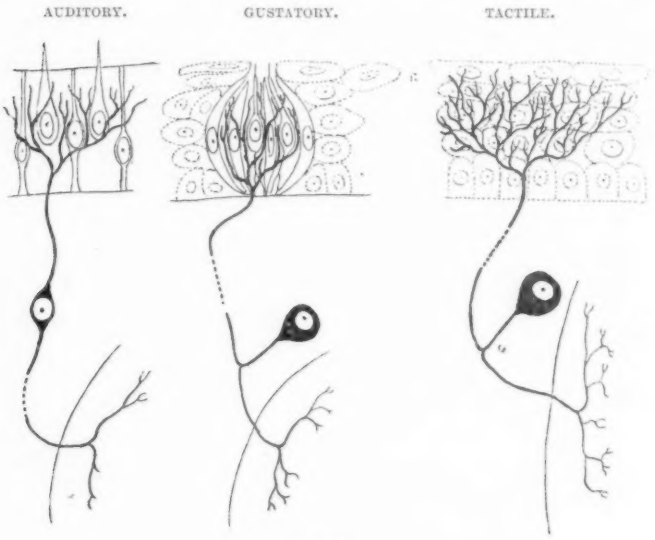


FIG. 16.

NERVE-CELLS OF SPECIAL SENSE. (Modified from G. Retzius).

supposed, emerge from the receptive hair-cells, but really originate from bi-polar or uni-polar cells which are placed somewhere in the course of the sensory nerve, and which resemble the cells which are found upon the spinal ganglia in sending a peripheral process to penetrate between the (somewhat modified) cells of the epithelium, and a central process to penetrate the grey matter of the nerve-centres.

In other sense-organs the nerve-cell itself more directly receives the sensory impression. This is so with the

rods and cones, or rather the rod-and-cone-cells of the retina and with the olfactory cells (fig. 17). In these cases the sensory impression appears to be received, if not directly by the body of the cell, at least by a very short process which is immediately attached to the body of the cell and which can scarcely be termed a nerve-fibre process, but is rather a specialised part of the cell-body; while from the other end

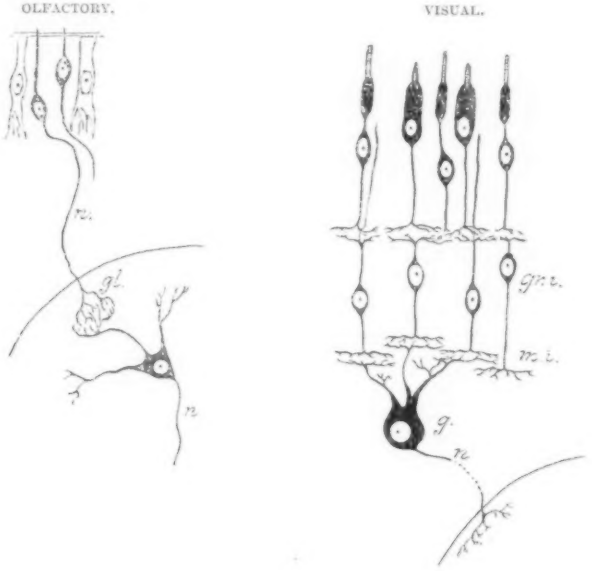


FIG. 17.

NERVE-CELLS OF SPECIAL SENSE. (Modified from G. Retzius).

n, *n*, neurons; *gl*, olfactory glomerulus; *g*, ganglion-cell of retina; *gr.i*, inner granules of retina; *m.i*, inner molecular layer of retina.

of the cell a fibre passes off which can only be regarded as a nerve-fibre and which ends, like other processes of nerve-cells, in an arborisation which becomes interlaced with the arborisations of other nerve-cell-processes, and thus allows the nerve-impulses which are generated within the sensory nerve-cells to be carried towards the nerve-centres. When one considers the differences met with in the several sense-

organs, viz.: that the sensations received through the skin and from the auditory and gustatory organs, are taken up by the terminal branchings of neurons, and that the sensations of light and of smell (which appear to depend upon more impalpable means of excitation), are taken up by the bodies of nerve-cells themselves, one is tempted at first to generalise from this to the effect that there is some essential difference between the two kinds of receptive organ, correlated with differences of function or of excitation. This generalisation will not, however, bear the test of further investigation, for we find from the observations of Lenhossek (19) and Retzius (32), that in the earth-worm all the sensory nerve-cells are wholly situated between the cells of the epidermis (fig. 14). There can be no doubt that these peripherally situated cells are nerve-cells, for like every other nerve-cell, each one has a neuron or nerve-fibre process (which in this case is directed immediately to the central ganglia, and ends by ramifying in the Punktsubstanz) and besides this, like many other nerve-cells, each cell has dendrons which ramify like roots underneath the epithelium but without anywhere joining the dendrons of neighbouring cells. These sensory cells are extremely numerous and are found over the whole epidermis, no other kind of nerve-termination or nerve-origin having hitherto been discovered within the integument. The nervous system of the earthworm is thus seen to be a perfectly diagrammatic structure (see fig. 14). Peripheral nerve-cells placed in the integument receive impressions and convert those impressions into nerve-impulses; the nerve-impulses are conveyed along afferent fibres to the ultimate ramification of those fibres within the Punktsubstanz, and from this Punktsubstanz they are collected either in the same form or more probably as new impulses by the ramifications of the collaterals of the neurons or neurodendrons of the motor cells and by these neurons are conveyed directly to the muscles. Now it is known that the earth-worm although it has no special sense organs, no eyes, no ears, no organs of olfaction, yet reacts to light, to sounds, to touch, and in all probability to odours. But all these impressions are operative upon the general integument and must be taken up by the

sensory cells within the integument and by these converted into nervous impressions. The generalization therefore which we might be apt to assume from the study of the sense-organs of vertebrates alone is thus seen to fall to the ground. Moreover Retzius has shown (35, 36) that in other worms (Nereis) and in mollusks the sensory cells tend to lie more deeply and are no longer placed entirely within the epidermis but beneath it. They then appear like bi-polar

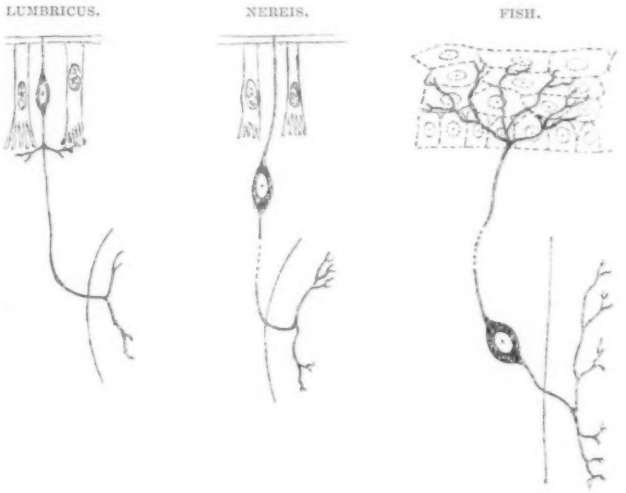


FIG. 18.

SENSORY NERVE-CELLS. (Modified from G. Retzius).

cells, sending a (relatively short) peripheral process to penetrate between the cells of the epidermis, and a long process centrally as a nerve-fibre which enters the ganglia of the central nervous system. Such cells offer a transition between the entirely peripheral or epidermal sensory cells of the earthworm, and the bi-polar cells of the spinal ganglia of osseous fish (fig. 18).

We may next consider how nervous impulses pass from nerve-cell to nerve-cell. We have seen that it is probable there is never actual continuity between nerve-cells, but only

contiguity of ramified processes, either with cell-bodies or processes of other cells. Physiologically it may be shown that there is always a partial block to the passage of a nervous impulse at the conjunction of one cell with another. This is evidenced by the loss of time which occurs when nervous impulses have to pass such a block. Thus there is such lost time at the motor projection-cells of the spinal cord; for if the time taken between the excitation of fibres of the pyramidal tract, and the commencement of the contraction of corresponding muscles be measured, it will be found to be prolonged (over and above the time taken by the transmission of nerve-impulses along the given length of nerve, *plus* the time taken by inertia of the muscle) to the extent of about .01 second (or less). This lost time which occurs at the junction of the cell-chain represents the period of latent excitation of the nerve-cell. Let us consider what is probably happening at this point. The nerve-fibre to which the excitation is applied carries nerve-impulses, which become spread out in the fine arborescence, which forms the termination of that fibre and which enwraps the motor-cell. From this cell nerve-impulses start, which are not necessarily of the same rate as those which have reached the terminal arborescence just mentioned, and these new nervous impulses pass down towards the muscle and cause its contraction. It is clear that a change occurs at the adjunction of the arborescence with the cell-body. A change in rhythm certainly occurs (16), and this renders it extremely probable that the nerve-impulses which are passing off from the spinal-cord-cell are entirely new impulses. If so, we may look upon this cell as having been freshly stimulated by the impulses which have passed along the fibre of the pyramidal tract. We may briefly consider in what manner it can be thus stimulated. Since there is no evidence that the fibrils of the arborescence anywhere touch the cell-body or its processes, we must assume that a space intervenes everywhere between the two, very narrow indeed but still a space which cannot readily, if at all, be traversed by nerve-impulses. It is possible to suppose that the nerve-impulses reach the pericellular arboresence, and produce by mere

induction new nervous impulses within the cell around which they play. But we have no evidence that such nerve-induction is possible. It is also open to us to suppose that the electrical change (action-current) which accompanies the passage of the nerve-impulses to the arborescence, may itself be the excitant of the nerve-cell, and that the nerve-cell may respond to this excitation by a rhythmic change of some kind, which is possibly contraction, possibly rhythmic chemical action, possibly molecular vibration, or perhaps a combination of two of these. At all events, it is probable that a new process is started within the nerve-cell. This does not necessarily follow from the fact that there is time lost at the adjunction, for a partial block to the passage of nerve-impulses and a resultant loss of time may be produced merely by mechanical means; but the change of rhythm renders it extremely probable. In considering this question, another one presents itself, viz.:—why do not nerve-impulses pass *backwards* from the motor cells of the cord to the fibres of the pyramidal tract: in other words, why are not nervous impulses generated in the terminal arboresence of the pyramidal tract-fibre, when an anterior root-fibre is stimulated? It is not a sufficient answer to this question to say one is the natural or habitual, and the other an an unnatural direction, for it has been shown by Gotch and Horsley (13)—to whom we owe our knowledge of the fact just mentioned—that nerve-impulses pass very readily from the nerve-centre *down the afferent channels*, although the strongest excitation will not cause them to pass from the efferent channels *up the spinal cord*. Since the effects pass both down and up the sensory paths, one would have expected that they would also pass both down and up the motor paths. In seeking for an explanation of this difference it has occurred to me that such an explanation may be found in the anatomical arrangement of the terminations of the pyramidal-tract-fibres around the motor cells as compared with the mode of central termination of the sensory fibres within the grey matter. It is conceivable that such structural arrangement may allow of the excitation of new nerve-impulses within the body of the

motor-cell, by an electrical discharge from the fine brush of pericellular fibrils which envelopes the body of the motor-cell, whilst the electrical change which accompanies nerve-impulses up the motor fibre, when this is artificially stimulated, may be so diffused throughout the cell-body of the motor-cell, as to fail to stimulate and set up nerve-impulses in the pericellular ramification of fibrils, which represents the ending of the fibre of the pyramidal tract.

From the present consideration of this subject we arrive at the following general conclusions:

1. That every nerve-cell forms a structural element which is anatomically isolated from but in physiological continuity with other nerve-cells.

2. That the physiological continuity of these elements depends on the contiguity either of ramified cell-processes of different nerve-cells with one another, or of the ramified processes of one cell with the body of another cell. By this contiguity such isolated anatomical elements become concatenated into nerve-chains which may be either simple, as in the nervous system of the earth-worm (fig. 14), and possibly in the ordinary reflex chain of vertebrates (fig. 15), or may be complicated to any imaginable extent (fig. 1).

3. That the same nerve-impulses do not necessarily pass from one element of a nerve-chain to the next, but that more probably new impulses (often of different rhythm) are generated in the successive elements of the chain.

4. That the new impulses so generated may be set going by the electrical change which accompanies the advent of the producing impulses and are not necessarily due to the passage of those impulses themselves to the next element of the chain.

5. That either the body of the cell or any of its processes may be concerned both with the starting and with the transmission of nerve-impulses; and, that these may originate by acts of contraction causing waves of pressure or variations of surface-tension to traverse the fibrils.

6. That the body of the cell is especially concerned with presiding over the nutrition of the whole cell-element; this trophic function being intimately associated with the presence of the nucleus. Nevertheless nerve-impulses may both origi-

nate in and be conducted by the cell-body. The dendrons or protoplasmic processes, being extensions of the protoplasm of the cell, may primarily serve to assist in the nutritive processes, as was supposed by Golgi, but they undoubtedly also, like the cell-body itself, may in some cases convey nerve-impulses.

7. That the ordinary centrifugal paths are blocked for centripetal impulses although the centripetal paths may convey centrifugal impulses, this physiological difference being correlated with a difference of anatomical relationship at the junction of the respective cell-elements.

BIBLIOGRAPHY.

(1) d'Arsonval, A. "Relation entre la tension superficielle et certains phénomènes électriques d'origine animale," *Archives de Physiologie*, 1889.

(2) Biedermann. "Ueber den Ursprung u. die Endigungsweise der Nerven in den Ganglien wirbelloser Thiere," *Jenaische Zeitschr. f. Naturwissensch.*, 1891.

(3) Edes, R. E. "On the method of transmission of the impulses in medullated fibres," *Journal of Physiology*, vol. xiii., 1892.

(4) Ehrlich, P. "Ueber die Methylenblaureaction der lebenden Nervensubstanz," *Deutsche medic. Wochenschrift*, 1886.

(5) Fritsch, G. "On the electric organs of Fishes," *Nature*, Jan. 19th, 1893.

(6) van Gehuchten, A. "La structure des centres nerveux," *La Cellule*, 1891.

(7) van Gehuchten, A. "Les cellules nerveuses sympathiques," &c., *La Cellule*, 1892.

(8) Gaskell, W. H. "On the relation, &c., together with a theory of the origin of the Nervous System of Vertebrata," *Journal of Physiology*, vol. x., 1889, p. 206.

(9) Golgi, C. "Recherches sur l'histologie des centres nerveux," *Archives italiennes de biologie*, 1883.

(10) Golgi, C. "La cellula nervosa motrice," *Archivio italiano per le malattie nervose*, 21, 1884.

(11) Golgi, C. "Studii sulla fina anatomia degli organi centrali del sistema nervoso," *Milano*, 1885-86.

(12) Golgi, C. "Ueber den feineren Bau des Rückenmarkes," *Anat. Anzeiger*, 1890.

(13) Gotch and Horsley. "On the Mammalian Nervous System, its functions and their localisation determined by a new method," *Phil. Trans.* vol. 182, B, 1891.

(14) His, W. "Die Neuroblasten u. deren Entstehung im embryonalen Mark," *Arch. f. Anatomie*, 1889.

(15) Hodge, C. F. "Some effects of electrically stimulating ganglion-cells," *American Journal of Psychology*, 1889 *and* 1892.

(16) Horsley and Schäfer. "On the character of the muscular contractions which are evoked by excitation of the various parts of the motor tract," *Journal of Physiology*, vol. vii., 1886.

(17) Kölliker. "Vortrag auf der Anatomenversammlung in München," *Verhandl. der Anat. Gesellschaft, Anat. Anzeiger*, 1891.

(18) Langley, J. N. & Anderson, H. K. "The action of nicotin on the ciliary ganglion," &c., *Journal of Physiology*, vol. 13, p. 465.

(19) von Lenhossék, M. "Ursprung, Verlauf und Endigung der sensibeln Nervenfasern bei Lumbricus," *Arch. für mikr. Anatomie*, Bd. 39, 1892.

(20) von Lenhossék, M. "Beobachtungen an den Spinalganglien und dem Rückenmark von Pristiurus-embryonen," *Anat. Anzeiger*, 1892.

(21) Leydig, F. "Zelle und Gewebe," *Bonn*, 1885.

(22) Magini, G. "Alcuni nuovi caratteri differenziali d. cellule nervose," *Atti. d. r. Accad. dei Lincei*, 1890.

(23) Marchi, V. "Sulle degener. consec. alla estirpazione totale e parziale del cerveletto," *Rivista sperim. di freniatria*, &c., 1888.

(24) Mosso, A. "The Temperature of the Brain, especially in Relation to Psychical activity," Croonian Lecture. *Phil. Trans.*, 1892.

(25) Nansen, F. "The structure and combination of the Histological Elements of the Central Nervous System," *Bergen*, 1887.

(26) Ramón y Cajal, S. "Sur la morphologie et les connexions des éléments de la retine des oiseaux," *Anatom. Anzeiger*, 1889.

(27) Ramón y Cajal, S. "Sur l'origine et les ramifications des fibres nerveuses de la moelle embryonnaire," *Anatomischer Anzeiger*, 1890.

(28) Ramón y Cajal, S. "Origen y terminación de las fibras nerviosas olfatorias," *Gaceta sanitaria de Barcelona*, 1890.

(29) Ramón y Cajal, S. "Sur la structure de l'écorce cérébrale de quelques mammifères," *La Cellule*, 1891.

(30) Ramón y Cajal, S. "Pequeñas contribuciones al conocimiento del sistema nervioso," *Barcelona*, 1891.

(31) Retzius, G. "Zur Kenntniss des Nervensystems der Crustaceen," *Biologische Untersuchungen, Neue Folge*, Bd. 1, 1890.

(32) Retzius, G. "Das Nervensystem der Lumbricinen," *Ibid*, iii., 1892.

(33) Retzius, G. "Die Endigungsweise des Gehörnerven," *Ibid*, 1892.

(34) Retzius, G. "Ueber den Typus der sympathischen Ganglienzellen der höheren Wirbelthiere," *Ibid*, 1892.

(35) Retzius, G. "Das sensible Nervensystem der Polychäten," *Ibid*, iv., 1892.

(36) Retzius, G. "Das sensible Nervensystem der Mollusken," *Ibid*, 1892.

(37) Retzius, G. "Die Nervenendigungen in dem Geschmacksorgan der Säugethiere und Amphibien," *Ibid*, 1892.

(38) Retzius, G. "Ueber die sensiblen Nervenendigungen in den Epithelien bei den Wirbelthieren." *Ibid*, 1892.

(39) Retzius, G. "Ueber die neuen Prinzipien in der Lehre von der Einrichtung des sensiblen Nervensystems, *Ibid*, 1892.

(40) ROHDE. "Histologische Untersuchungen über das Nervensystem der Polychäten," *Breslau*, 1887.

(41) ROHDE. "Histologische Untersuchungen über das Nervensystem der Hirudineen," *Breslau*, 1891.

(42) ROLLESTON, H. D. "On the Conditions of Temperature in Nerves (i) during activity; (ii) during the process of dying," *Journal of Physiology*, vol. 11, 1890.

(43) SCHÄFER, E. A. "Observations on the Nervous System of Aurelia aurita," *Phil. Trans.*, 1877.

(44) VERWORN, M. "Die physiologische Bedeutung des Zellkerns," *Pflüger's Archiv.*, Bd. 51, 1891.

(45) WALDEYER, W. "Ueber einige neuere Forschungen im Gebiete der Anatomie d. Centralnervensystems," *Deutsche med. Wochenschrift*, 1891.

ON THE VISUAL PATH AND CENTRE.[1]

BY PROFESSOR S. E. HENSCHEN, M.D., OF UPSALA, SWEDEN.

BEFORE an audience like this, I certainly need not enlarge upon the great importance of the senses in the development of our intelligence, and indeed of our whole psychical life, of which doubtless the perceptions through our senses are the necessary conditions and basis. In order that modern psychology may possess a definite starting point and foundation, we must obtain an exact idea of the anatomical construction and physiological function of the paths and cortical areas of our senses, as well as their connections with other parts of the brain. In consequence of the law of evolution, there exists in man the highest development of the different centres, and of psychical life. For this reason we must principally study the paths and centres of the senses in *him*, whilst comparing the results obtained by experiments on animals.

For the deeper study of the senses, there is none so instructive as that of sight, which is, in certain respects, the principal one. Through the same we procure the greatest wealth of perceptions, which will continually serve as objects of our psychical activity. No sense can be so minutely examined in regard to its functions and pathological derangements, and a clinical examination will, in most cases, determine whether the lesion be of a peripheral or central nature. If we are ever to grasp the inner connection between the higher psychical activity and the construction and function of the senses, it must be principally through the deeper study of the visual path, its centre and activity. The more profound and penetrating the knowledge we obtain in this direction, the more certain will be the starting point of our

[1] Paper read before the Congress of Experimental Physiology in London, 1892.

psychological researches, not only in this direction, but also in that of the other senses, for we may presume that there exists a certain analogy between them. It is for this reason I take the liberty of giving a short sketch of my opinions on the above, viz. :—the visual path and centre, based upon clinical facts gathered from others as well as myself. But I shall not enter into details, and the prescribed time will not allow me to bring forward the proof for many of my views, but for further particulars I refer you to my work, "Beiträge zur Pathologie des Gehirns." The second volume has just now been published, a copy of which I now have the honour of laying before this congress.

In order to understand the nature of the cerebral representation of vision and its psychology, the localisation of the cortical centre for sight, is of more interest than that of the course of the visual path, which is principally an anatomical question. But the former proposition cannot be solved without a knowledge of the latter, and I shall, therefore, first discuss the question of the visual path. Moreover for a proper comprehension of the relation of the visual centre with those of other senses, it is necessary to have an acquaintance of the connection of the visual path with the central ganglia of the brain. I will, then, in considering these anatomical questions, express myself briefly, and mainly dwell upon some of the most important points, in which I differ from current doctrines.

Enquirers have tried in different ways to arrive at a solution of the question of the visual centre. Wilbrand, for example, has by using the purely clinico-ophthalmological method elaborated a theory, which may not be unknown to my auditors. Objections might be raised against his conclusions, in that the starting point of his statements is uncertain, in this respect, that only a few of his ophthalmological cases were examined after death. Nevertheless, I have, by using the combined clinico-anatomical method, arrived at much the same conclusions. Only by this latter method, the clinico-anatomical, can we secure a firm basis for the investigation of this field of study. Time after time, it has been adopted by celebrated observers, such as Ferrier, Luciani,

Tamburini, Wilbrand, Starr, Seguin, Nothnagel, Seppilli, &c., but the results at which they arrived have only partly coincided. Time will not suffice to give an historical sketch of their different opinions. I will only intimate that whilst some Frenchmen wish to extend the visual centre as far as the central convolutions, others, especially Englishmen, limit it to the occipito-angular region, and others, as Seguin and German authorities, confine it to the occipital lobe. Reinhardt would place the centre, in agreement with Munk's experiments, on the lateral surface of the occipital lobe, Nothnagel in the first occipital gyrus, Seguin on the mesial surface, and Hun places it immediately in the neighbourhood of the calcarine fissure. A new and rigorous analysis of the clinico-anatomical cases is of course necessary, and I have endeavoured to submit to it all accessible facts concerning the visual centre, and I am convinced that all these tend in the direction I propose indicating.

Besides these clinico-anatomical facts, there exists a number of other pathological ones of importance in the solution of the above problem. After extirpation of the bulbs, or a lesion of the frontal visual path, or of the external geniculate body, there arise centripetal degenerations; and after lesions of the visual cortex, arise degenerations of a centrifugal nature, which enlarge our knowledge of the centre itself. These changes have been principally studied by Monakow and myself.

Numerous investigators have devoted much time and patience in experiments upon animals, but I omit stating their conclusions here, in the belief that these renowned experiments are known by all present. Meanwhile, one should, with great circumspection only, be permitted to apply the results of experiments upon animals directly to man, especially as experimentors have not arrived at the same results.

In the first place, in order to discover the visual path and centre, every clinical case supplemented with a *post-mortem* is serviceable, in which some defect of central origin is observable in the field of vision. These defects are almost always hemianopic. But nearly as important as the positive cases are the negative, with an absence of any defect in

the field of vision, but with a lesion in the neighbourhood of the visual path or centre. These latter control the conclusions drawn from the positive cases. A necessary condition, to obtain a sure result, is that the clinical and pathological examinations are conducted by competent persons, because hemianopsia is often latent and frequently impossible to detect in feeble and unintelligent patients. Since I commenced to make a systematic examination of my patients in this respect, I have found in my hospital in Upsala, nearly 40 hemianopic cases, supplemented in the *post-mortem* records; whilst Seguin, in 1886, could only discover about the same number in the whole of literature. The number of cases has considerably increased of late years, and there are now perhaps more than 160 on record. Lesions of the visual path, especially cortical ones, are also evidently very common, and we may hope that further clinical researches upon this matter will thoroughly elucidate the organisation of this sense. This, however, can only be the case, if clinical and pathological scientists work methodically. An analysis of the already published cases proves that the present manner of working does not satisfy the requirements of science, for many even of the most important cases are incomplete. My statements are chiefly based upon my own cases.

The Visual Path can be divided into the frontal, middle and occipital. I shall not dwell upon the anatomical construction of the frontal visual path, as it is only of minor importance in elucidating the problem before us. Nature has herself indicated its direction. In the optic nerves, chiasma and tracts, the fibre from different quadrants lie in separate bundles, and those from corresponding points are not immediately beside each other. Fibres from the upper retinal quadrant lie dorsally in the tracts.

The Middle Path.—The course of the optic fibres and their connection with the central ganglia has not yet been thoroughly investigated. It is very important to notice that it is necessary to distinguish between visual and optic fibres; a lesion of the former produces a defect in the visual field alone, whilst the latter are to be considered as reflex

fibres. There is no doubt that the three great central ganglia, the external geniculate body, the pulvinar and the corpora quadrigemina anterior, all receive fibres from the optic tracts; but the question is, do they all receive visual fibres? Thereupon depend their several functions. The above three main ganglia have evidently different duties to perform in different animals, but I only here intend referring to their importance in man. The solution of this problem should be effected by an analysis of the respective clinico-pathological cases. The central ganglia are often injured by lesions, which are succeeded by hemianopsia, but these are often microscopical, and extend simultaneously with larger lesions into the occipital or parietal lobe, and, of course, are then overlooked.

Literature specifies at least eight[1] cases of tumour in the central ganglia, six of hæmorrhage, and sixteen of softening, accompanied with hemianopsia. An analysis of these cases gives the following results. In the case of tumour the growth was so diffuse that no enlightenment could be obtained concerning the functions of the above three main ganglia. Of the cases of hæmorrhage there were only two, Dreschfield's and my own, in which the lesion was so circumscribed that a conclusion could be drawn of the function of the pulvinar, but from the freshness of the hæmorrhage and its pressure upon neighbouring ganglia the result could but be considered uncertain. Neither are the sixteen cases of softening of use in the solution of the problem, and this for reasons you will find discussed in my book. Thus the analysis of the clinical cases with hemianopsia does not decide this question, and here, therefore, the importance of the negative cases arises. Nothnagel has, by an analysis of lesions in the corpora quadrigemina, proved that such need not give rise to any form of visual disturbance, and further, some cases go to prove that the destruction of the pulvinar is not always accompanied with hemianopsia. By means of exclusion we shall also prove that there is no clinical proof that in man the pulvinar or corpora quadrigemina ought to contain visual fibres and thus be necessary to

[1] See my work "Pathologie des Gehirns," Upsala, 1892, p. 266.

sight. Consequently, the external geniculate body is the main sight ganglia in man. Its destruction will always produce some form of hemianopsia.

The position of the fibres in this ganglia has only partially been ascertained. My own researches in a case of atrophy of one optic nerve have persuaded me that fibres from both eyes lie immediately together in this ganglia, and not in separate bundles, as in the frontal portion of the visual path. And, finally, Monakow has on account of his own enquiries expressed the opinion that the visual fibres do not lie in direct connection with the ganglia cells, but ramify in the ground substance. To the above I will only add that the clinical analysis does not render any support to the commonly accepted theory that a lesion in the posterior portion of the internal capsule gives rise to hemianopsia, and still less to the idea that a decussation in the middle line of the corpora quadrigemina takes place.

The Occipital Visual Path.—The determination of the situation and limits of the occipital visual path is of the greatest moment in deciding the debated question as to whether the parietal, and especially the angular gyrus, belongs to the sight area or not. It is very common for lesions in the parieto-angular region to be accompanied with hemianopsia, and this fact has given rise to the opinion that it belongs to the sight area. But a critical analysis of the cases tends towards another view, for in a considerable number of cases lesions in that region are not followed by hemianopsia. How, then, can we find a clue to this apparent contradiction? The explanation is as follows:—If the lesion implicates the visual path in the optic radiation of Gratiolet, it will induce hemianopsia, but not otherwise.

Where does the Visual Path lie?—This question will be solved by a clinico-pathological analysis. Literature mentions at least 13 cases of abscess or tumour in the parietal lobe. In eight of these hemianopsia was present; in five not. There are also four traumatic cases. In most of these the lesions (tumours or traumata) were so large and diffuse, or they pressed upon the optic radiation to such an extent, that their influence cannot be accurately determined. The negative

cases are, again, of decisive importance, for they prove that a lesion of the parieto-angular cortex need not induce a defect in the field of vision. It is just the same in cases of hæmorrhage. Six were followed by hemianopsia, but in one of mine—that of a professor of medicine—the hæmorrhage was not accompanied with hemianopsia. Finally, there exist at least 25 cases of softening in the parietal lobe, in 12 of which hemianopsia was present; in seven absent. An analysis of all these cases gives us this result: the visual path is situated in the ventral portion of the optic radiation, and there forms a bundle less than a centimètre thick, which lies at the level of the second temporal gyrus and second temporal sulcus. A lesion of the parietal or angular lobes induces hemianopsia, only if this bundle be pressed upon directly or indirectly, a lesion of the dorsal part of the optic radiation does not produce a defect in the field of vision. One of Dr. Anderson's, and another of Dr. Bruns' cases, show in what manner the fibres lie in the bundle forming the visual path. The fibres for the dorsal retinal quadrant lie dorsally, and one of my own cases shows that the fibres for the ventral retinal quadrant lies ventrally. It is probable that the macular fibres have a more median situation than the peripheral.

We now enter upon the consideration of the occipital lobe. A number of observations have been made in literature upon occipital lesions. Most of them were accompanied with hemianopsia, but only a few of them are of use in the accurate study of the localisation of the centre of vision. In 20 cases the lesion was diffuse in the medulla of the occipital lobe. None of them localise the visual centre to a circumscribed portion of the lobe, because the visual path was destroyed by the lesion, but many of them go to prove that it does not extend further than the occipital lobe. All cortical or sub-cortical circumscribed lesions, on the contrary, are of great importance in the localisation of the visual centre. The first question that arises is, Does the centre of vision lie on the lateral, mesial, or ventral surface? Eight cases with cortical lesions on both the lateral and mesial aspects of the cortex do not solve this question, but

the fact that in 11 cases of lesions situated in the lateral centre, there existed no defect, or only a transitory one, in the field of vision, proves that we need not seek the visual centre in that situation, more especially as, in some cases, the lesion was bilateral. On chart H in my book, I have collected a number of cases without hemianopsia, but with lesions on the lateral or ventral surface of the parietal or occipital lobes, and a look at it ought to convince us that we need not expect to find the visual centre in either of these regions. Just as convincing are the cases with lesions exclusively on the mesial surface (chart G), of which literature mentions at least 25 examples. A methodical analysis of them leads to the following results :—Firstly, a lesion on the mesial surface causes hemianopsia only if the cortex of the calcarine fissure, of the fibres derived from it, are affected. Secondly, a lesion limited to the calcarine cortex can induce a complete hemianopsia, and of this the most instructive case is, I believe, one now published in my book, which includes all guarantees for an exact conclusion. It was stationary, uncomplicated, and the clinical examination, as well as the *post-mortem*, was accurate. The lesion was limited to the cortex in the depth of the calcarine fissure, and the hemianopsia was complete and absolute. Besides, many other negative cases confirm this result, and, in particular, one of my own, with a bilateral destruction of the margo falcata, without defect in the field of vision, proves that the visual centre is not to be looked for there, as Nothnagel and others have affirmed. As to the still more accurate limitation of the visual centre in the cortex of the calcarine fissure, the cases, on the whole, are not sufficiently definite to decide, but there are not at present any absolute reasons for extending it further than the lips of the fissure. In some cases a complete hemianopsia resulted without a lesion in the frontal part of this cortex ; in others without any affection of the most posterior part ; but in all cases the middle part of the fissure was implicated.

I cannot find in literature any one well-described cases which stands in opposition to the opinion here expressed ; but there are many appearing to lead to other conclusions, but which cannot stand a rigorous criticism. Monakow has

quite recently arrived at a contrary result. I will not in his absence criticise his theory on a variable localisation of the macula latea, but will only point out my own individual stand-point. I cannot find the clinical cases on which Monakow bases his opinion. He suggests that many spots in the parieto-occipital cortex can, under different circumstances, represent the field of vision. The way in which the fibres of the visual path, pass through the occipital lobe to reach the calcarine cortex, is not clear, but they probably lie in both the dorsal and ventral aspects of the optic radiation. Several facts go to show that, at least, the cortex for the macular field in the calcarine fissure is innervated in a double way.

Organization of the Centre of Vision.—The next question is: How is the centre of vision organised? At first there exists a projection in the calcarine fissure. I have mentioned above that my researches have led me to the conclusion that the fibres of the dorsal retinal quadrant lie dorsally both in the frontal and occipital visual path. The interesting case of Hun's, proves that there is the same arrangement in the calcarine cortex. Thus the upper lip represents the upper retinal quadrant, and other cases strengthen this opinion.

The question now arises: Where does the macular field lie in the calcarine cortex? In deciding this, there are no positive facts, but many cases indicate that the macular field may be more anterior, and the peripheral in the horizontal meridian, more posterior. Some time ago, I observed a case in which the whole of the right occipital lobe was destroyed, and the tip of the left damaged, but still the patient could read up to her death. This case is against the localisation of the macular field being at the tip of the occipital lobe.

Perhaps it is known that Wilbrand, by using purely clinico-ophthalmological facts has come to the conclusion that the macula lutea is innervated by both hemispheres in a variable manner. The clinical analysis of the cases seems to confirm this opinion, and such an arrangement is very favourable to those unfortunate enough to be afflicted with

hemianopsia, for they will still preserve the faculty of sight at the point of fixation. As above stated there seems to be another guarantee for the preservation of the macula lutea, and that is, double innervation, both from the dorsal and ventral portions of the optic radiation.

The third important question concerning the cortical retina, as I should like to call the cortex of the calcarine fissure, is whether both eyes or only one are represented in the lips of the fissure, and the crossed as well as the uncrossed fibres. This question is answered by the above case, where a lesion limited to the calcarine fissure had induced a complete hemianopsia of both sides, and others of my cases tend to confirm such a conviction, although it is in opposition to Munk's celebrated experiments.

Of as great interest is the question as to whether the same elements represent homologous points of the retina, or not, and how they are situated in relation to each other. I have myself observed some important facts bearing upon this question, and Wilbrand has elaborated a theory on the subject, supported by some clinical facts. After destruction of both eyes in a patient suffering from leprosy, there ensued a complete atrophy of the calcarine cortex, but in a case of destruction of one eye only, I found in that situation, a number of pigment-charged cells alongside of perfectly normal ones. These facts seem to prove that the elements of both retinal halves are represented in the calcarine cortex by different cells, which lie beside each other. As to the perception of colours, the commonly received opinion that it is situated on the ventral surface will not admit of criticism. Some of my own cases prove positively that colour perception is also situated in the calcarine cortex.

It is well known that this cortex is characterised by a special anatomical construction, and particularly by solitary cells and a layer of small cells, corresponding with the streak of Vicq d'Azyr.[1]

The above expressed opinion in regard to the organisation and limitation of the visual centre to the calcarine fissure does not stand in opposition to the results obtained by the

[1] See my work, p. 378.

investigation of centripetal and centrifugal degenerations. Monakow also has found by this method that the calcarine fissure stands in connection with certain groups of cells in the external geniculate body. I will not discuss here the complicated problem as to which cells receive the impression of light. It might be left an open question, as well as that of the functions of the different layers in the cortex. After the destruction of the bulbs, or of the external geniculate body, the large as well as the small pyramidal cells undergo essential changes. If the above theory resists criticism, then many of the main problems of the visual path are solved. But we must further exert ourselves to seek new observations, still more accurate and detailed than those hitherto discovered, before the above theory can be considered as finally settled.

The next fundamental problem will be to discover the functions of the remainder of the occipital cortex and angular gyrus. But there is no doubt that this cortex has a certain connection with the function of the eyes. Word-blindness, and some other facts in human pathology corroborate such an opinion. I will not comment upon this very complicated problem, which is not yet, in my opinion, ripe for solution.

All these problems are of the greatest importance to psychology, but they cannot be decided without having obtained a solution of the visual centre. Therefore, it seems to me that this study is the most serviceable to us in indicating the right way leading from physiology to psychology.

ON THE PSYCHICAL NATURE OF HYSTERICAL UNILATERAL AMBLYOPIA AND SENSITIVO-SENSORIAL HEMIANÆSTHESIA.

BY PROF. BERNHEIM (NANCY).

At the Congress for the Advancement of Science held at Nancy in 1886, I first demonstrated that hysterical unilateral amblyopia behaves exactly like suggested amblyopia; it is purely psychical. The subject sees with his retina, sees with his brain: the first receives the impression, the second perceives it by its visual cortical centre. But this perceived visual image is unconsciously neutralised by the mind of the subjects of hysterical or suggested amblyopia. They see with the bodily, not with the mental, eye; their amaurosis is but a negative illusion.

I demonstrated this fact by means of a prism and of Snellen's apparatus. We know that a prism placed in front of an eye displaces the corresponding image, and thus produces diplopia. The other eye being closed, there is no diplopia, and the object seen through the prism appears single.

In unilateral amblyopia, hysterical or suggested, a prism placed before the healthy eye should give but a single image, the other eye being supposed to be blind. Now, the patients (who are unacquainted with the theory of the prism) do not hesitate to say that they see two equally definite images, corresponding to the disposition of the prism; hence the amaurotic eye does see.

Stoeben's modification of Snellen's apparatus consists of a pair of spectacles, with a green and a blue glass, and of a black frame, on which there are six letters alternately covered with red and green glass. When one looks to this

frame through the spectacles with both eyes open, the six letters are visible; when with one eye only, only three letters are seen, those of the same colour as that of the corresponding glass of the spectacles; the green letters with the green glass, the red with the red. This results from the fact that the mixture of red and green appears black.

Now, our patients, amblyopic through hysteria or suggestion, read all the six letters without hesitation; hence they see with the affected eye; when they close it they see three letters only.

I have shown that hysterical or suggested dyschromatopsia is also purely psychical. Here is a patient with dyschromatopsia of the left eye; a square of green paper are seen grey with it. Now the same square, when looked at with a prism before the right eye, appears double, hence the image formed by the affected eye is green.

Dr. Parinaud, who had first made this experiment, explained it by admitting that achromatopsia existed only in *monocular* vision. He supposes that, in looking with each eye separately, we connect the retina with the opposite hemisphere, but that in binocular vision each eye is connected with both cerebral valves. He has overlooked the fact that the phenomenon is purely psychical. Hysterical achromatopsia is merely a mental illusion concerning the colour of a seen object. The sensorial centre sees the true colour; the mind of the patient does not perceive it.

My explanation has been misunderstood, and it is why I return to the subject. Prof. Pitres, of Bordeaux, after repeating my experiment, says:—"I do not understand how a hysteric can unconsciously mentally neutralise monocular perceptions, without also neutralising binocular perceptions, or, at least, that portion of them supplied by the affected eye. The hypothesis of multiple sensory centres is more satisfactory to my mind, and until further reasons are given, I give it the preference."

My explanation will be clearer if I say that the patient's imagination falsifies or neutralises involuntarily and in good faith the perceptions of one eye, and the following case, in confirmation of this explanation, will disprove the opposed hypothesis.

A youth of 19 fell ill on the 6th of January with gastro-intestinal influenza; diarrhœa and vomiting lasted two days. At the same time there were fits of coughing, ending in attacks of stabbing pains on the left side of the face and body. He also had pain from the xiphoid cartilage to the throat. On admittance into the hospital, the patient, delicate, nervous, indifferently intelligent, was unaware that he was nearly completely hemianæsthetic on the left upper half of the body. The anterior aspect of the thigh alone was anæsthetic and analgesic in the lower limb.

With reference to this hemianæsthesia, let us first examine the amblyopia. The patient cannot read even large print with the left eye, and but vaguely recognises objects. A watch or a hand placed in front of him appear as dark surfaces. He does not see a finger before his eye; when there is nothing, he thinks he can see two fingers. There is also dyschromatopsia. On the 9th of January, white and blue are called red; yellow, blue; and red, grey. On the 12th white is black, and black white; blue is yellow, yellow blue, red grey. These discrepancies in colour vision lead one to assume that the imagination plays a part in the results obtained. The experiment of the prism placed before the right eye shows that in this case also the amblyopic eye sees, for two images of equal clearness are perceived in the respective situations following the disposition of the prism. When the left eye is closed, a single image is seen through the prism by the right eye.

We might suppose with Dr. Parinaud that left amblyopia exists only in monocular vision, but the following experiments disprove this hypothesis. With Snellen's apparatus, the two eyes are armed with red and green glass; the patient sees the six letters and recognises their colours. On closing the right eye he sees everything black. On closing the left eye (red glass), he sees only the green letters, corresponding to the vision of the right eye. This result shows that the left eye sees colours and objects, since he can read the letters without binocular vision, since the right eye cannot see the letters perceived by the left eye only.

One might suppose, it is true, that opening the two

eyes is enough to allow each eye to enter into communication with both hemispheres, and that thus the left eye could see with the left hemisphere; but the following experiments show this supposition to be erroneous. After ascertaining that with both eyes open he sees all the letters and their respective colours, that with the right eye only he still sees them all, but perceives nothing with the left eye, he puts on the spectacles. With the right eye (red glass) alone, he reads the red letters; with the left (green glass), he reads the green ones, but does not recognise the colour. Hence the left eye sees objects in thonocular vision, through the right hemisphere, without assistance from the other eye. It sees when the patient's imagination is led astray, and cannot intervene to neutralise or falsify perception.

The following experiment is still more conclusive:—On the 18th of January I send the patient for examination to the eye department. Dr. Koehler, after ascertaining that the left eye cannot read nor recognise objects, says that he is going to examine for selecting glasses, and, with this view, tests the visual acuity by means of Monoyer's scale. He finds that for both eyes it is equal to 0.6; for the right eye, to 0.67; for the left, to 0.5. The left eye alone easily reads the last five lines of the scale.

I repeated the experiment on the 19th, as I could not understand how the patient who was unable to read the largest print on a book or a newspaper had been able to make out much smaller type on the scale. To my great surprise, at a distance of even five metres, he could easily read the five last lines, and clearly distinguish the black letters on a white ground, but at a less distance than five paces he ceases to distinguish, and sees only white strokes on a black ground.

I then try him with colours. Black is seen white (with the left eye) as far as five paces; beyond it appears as black. Like white, blue, and red are seen grey within the limit, but are distinguished beyond. This true vision of objects and colours does not develop gradually, but becomes abruptly manifest at the distance of five paces. The colours and letters of Snellen's apparatus are seen with the naked left

eye at five paces and beyond. Within this distance all is black and undefined. With a prism, the left eye alone sees colours at three paces already; the patient, led astray by the prism, has badly calculated the distance; a blue book then is seen blue, but grey at a shorter distance.

These contradictory results can be explained only by the effects of imagination. The mind of the subject, disturbed by the experimental methods, in presence of Monoyer's scale, omits to inhibit the perceived images and colours; and thus the auto-suggestion arose, to see at the distance of five paces. Once surprised into seeing correctly, the eye has kept up the tradition, as it were, and persisted in seeing—but only at the same distance.

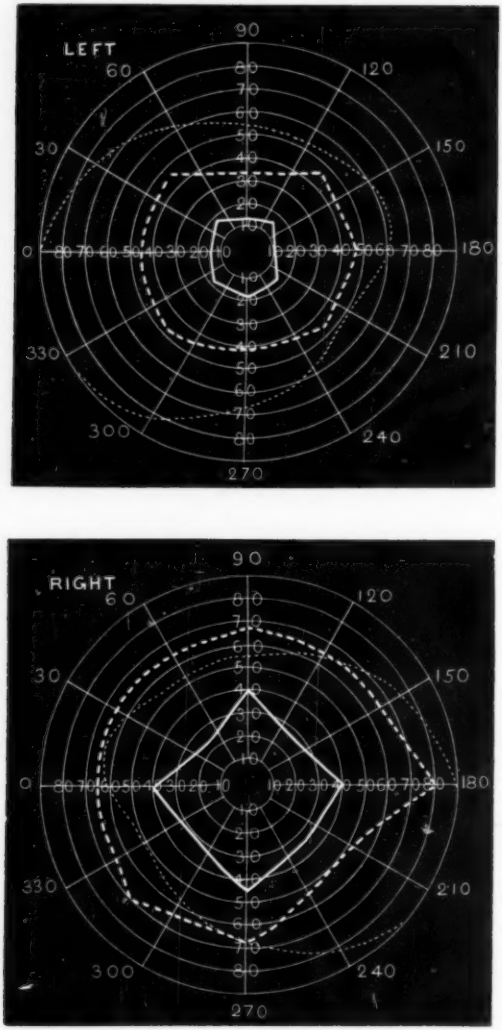
All this certainly looks very like a gross imposition. Is it simulation? Yes, if you like; but it is unwitting, compulsory simulation, like that of an hypnotized person.

Both visual fields[1] of the patient are contracted, much more so on the left side. This is the rule in hysteria. The tracing cannot be simulated in a subject like ours, who is quite ignorant of medical matters. He has never been in a hospital, and ignores that hysteria produces such unequal contracted fields of vision. Moreover all hysterical and suggested amblyopias, when examined with prism and Snellen's apparatus, behave like that of our subject. Others, it is true, are more logical and intelligent, do not allow themselves to be too easily taken by surprise, and do not betray the auto-suggestive or pseudo-simulating nature of the phenomenon. But in all it can be shown that the amblyopia appears to be simulated. But then, must we admit that all hysterics combine for shamming unilateral amblyopia, contracted fields, and all the other symptoms of hemianæsthesia?

Let us examine this symptom in our lad. It appears to be complete, tactile, analgesic, thermic, muscular. Pricking, pinching, electrising the left side have no effect. And yet the patient has little self-control; the same stimuli applied to the right side call forth lively demonstrations of pain.

Even when taken unawares the unexpected prick of a

[1] See figs. 1 and 2.



FIGS. 1, 2.

The plain line. Contraction of visual fields on Jan. 10th.
The dotted line. State of visual fields on Jan. 24th, after a suggestion of cure.

pin on the left side leaves him absolutely unmoved. Moreover, the left hand and forearm are bluish and cold, showing the existence of something objective. Some experiments, however, show that there are psychical elements in the mechanism of this anæsthesia. Our patient has thermic anæsthesia, and paræsthesia; a cold object applied to the left hand or forearm appears to be warm, and a warm object cold. On the 15th January I apply his hand to a heated stove; he says it is cold, and leaves his hand in contact, to the extent of burning it, but without manifesting any pain. On opening his eyes and seeing his hand, he says that he feels nothing, neither heat or cold.

This perversion of sensation is possibly but a psychical phenomenon; no physiological explanation can be given. Perverted imagination alone can suggest cold for warm—likewise white for black, *et vice versa*.

Muscular anæsthesia may also be shown to rest on a mental basis. The patient seems to have lost the notion of the position of his left arm. He cannot pick up a pin with it, nor say whether it is in the air or resting on the bed. With his eyes closed he cannot find his left hand with his right. My own hand placed in it is mistaken for his own right. The patient is hypnotized, and suggestion is made that his right hand is a magnet attracting the left. The suggestion is realised, and this hand goes to be applied to its fellow, showing that the apparent absent notion of its position does exist. After several successful repetitions I explain to the spectators that this experiment shows that the patient feels his hand. Now the experiment fails to succeed. What I have said sets up an auto-suggestion, and the muscular sense is again neutralized. The left hand is still attracted *towards* the right, but ceases to reach it. The patient is roused; he still cannot find his left, and mistakes mine for his. But if I place his own left into his right hand he rejects it as not being his own. He thus was proved to be guilty not of shamming but of auto-suggestion. With reference to auditory anæsthesia, it appears to be complete. He does not hear a watch close to the left ear. One day, however, whilst talking to him, I close his right ear, and

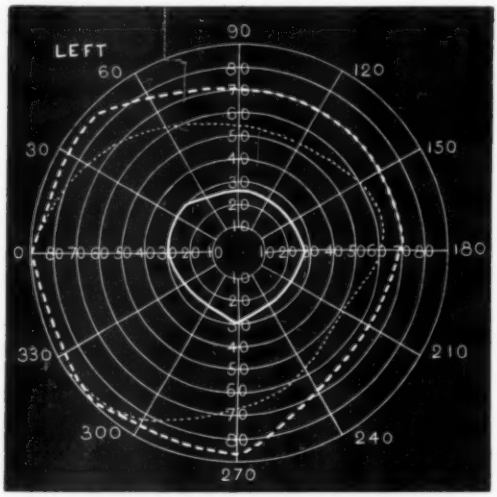
continue, quite naturally, to talk to him with a low voice, and from a certain distance. He answers quite well. On being asked how it is that he now hears, he answers that he does not know, that he seems to understand. But from this moment, his attention having been aroused, inhibition sets up, and he ceases to hear the loudest voice. Nay, I then free the right ear and continue to speak in high tones, but into his left ear, as if I was talking for it alone. He does not hear, though the right ear must perceive the sounds. He affirms that he hears nothing. I then talk to his right ear, and he answers. Here again he is surprised, as a victim of auto-suggestion.

The same applies to olfactory anæsthesia. A piece of paper dipped in vinegar and introduced into his left nostril causes neither sensation nor re-action. I push the paper backwards into the pharynx, so that the acetic vapour must affect the right nasal fossa. He continues to feel nothing, though the functions of the latter be otherwise intact.

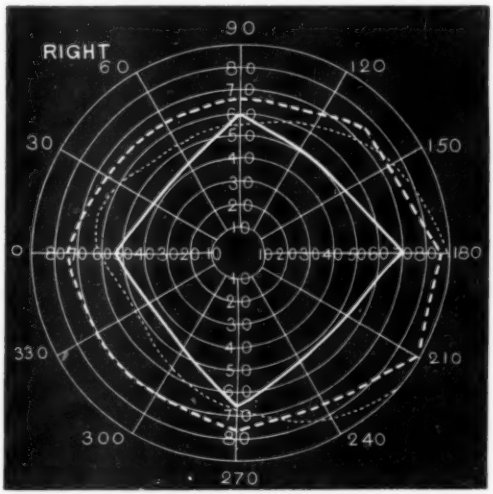
These experiments prove that in our subject, amblyopia, deafness, anosmia, akinæsthesis, are all purely of psychical origin. Hence, a psychical treatment against the tenacious auto-suggestivity of the patient, gradually restored, in three weeks, all the functions. I allowed one symptom to persist, viz.: the analgesia of the nape of the neck, which still subsisted three months later.

All these anæsthesias, let me repeat again, behave exactly like those due to hypnotic suggestion. But it may be said that hysterical amblyopia presents a great contraction of the visual field, on the diseased, a lesser one on the sound, side. Now I have found it to be the case in unilateral suggested amblyopia. Suggestion made during hypnotism of want of vision in one eye is followed with considerable contraction of the field on that side, and a lesser contraction of the other field. This is shown by the accompanying tracings (figs. 3 and 4).

I do not wish to prove thereby that no organic substratum underlies hysterical anæsthesia. How could a hysteric create by his imagination a left-sided hemianæsthesia? No subject can suggest to himself phenomena of which he has



The plain line. Left visual field after suggestion of amblyopia in this eye
The dotted line. Left visual field after suggestion of cure.



FIGS. 3, 4.

The plain line. Right visual field after suggestion of amblyopia in left eye.
The dotted line. Right visual field after suggestion of cure.

no idea. There must be in his right hemisphere something to set up the symptom. But this something is not in the cortical memory centres. We have seen that they perceive. It must be in the psychical centres, in the cells which are the seat of reaction in consciousness. These cells being modified, it is consciousness of impressions received, which is modified or neutralized. Some disturbance prevents their accepting, or makes them distort, the sensorial impressions from the opposite side of the body. It is a negative illusion, a mental alienation limited to sensations. It is a disease of the conscious æsthesodic cells of the cerebral hemisphere.

ON THE PRODUCTION OF SHADOW AND PERSPECTIVE EFFECTS BY DIFFERENCE OF COLOUR.

BY PROFESSOR EINTHOVEN (OF LEYDEN).

DIFFERENCE of colour may, under certain circumstances, be the cause of an apparent difference in distance.[1] To observe the phenomenon, it is only necessary to glue different coloured figures, such as letters of blue and of red paper, to a screen of black velvet, and to look at them from a suitable distance. In the experiment about to be described, Roman capital letters of about eight by four centimeters were used, the screen being placed at about three metres distance from the observer.[2]

Under these conditions it appeared, both to the writer and to others who he interrogated, that the red letters were nearer than the blue. Obviously, the phenomenon might be explained by difference in accommodation. In order to see the red letters distinctly, a greater amount of accommodation is necessary than in focussing the blue ones, and the greater sense of effort might account for the notion of the red letters being nearer. This accommodation hypothesis, plausible as it seems, cannot however be accepted as a satisfactory explanation of the phenomenon. Several observations tell against it, notably this: that there are about as many persons who see the blue letters before the red, as there are those who see red before blue. In the second place, the apparent difference in distance—so distinct to binocular vision—disappears almost wholly with the closure

[1] Donders. *Weteusch. bijbladen. Med. Gasth. v. Ooglijders*, 1868.

[2] W. Einthoven. "Stéréoscopie dépendant d'une différence de couleur." *Archives Néerlandaises*, T. 20.

of one eye. Looked at with one eye only, and for some length of time, the letters appeared to be lying in the same plane, but each time that the other eye was opened, the difference in distance obtruded itself irresistibly.

The amount of difference remains constant, and can be estimated with considerable accuracy, in the same way as in making a stereoscopical observation. The question therefore suggested itself, whether we had not here to deal with real stereoscopy? The answer to this question is an affirmative. Brücke[1] has shown by means of a simple experiment that the retinal images of differently coloured points are shifted with respect to one another. Looking with one eye at a narrow vertical strip on a black background, the upper and lower thirds of the strip being red and the middle third blue; Brücke observed that the blue part deviated to one side, the two red parts to the other side. By covering either eye alternately, a deviation of the blue and red parts in opposite directions will be observed; and, on both eyes being used, the notion of a difference in distance is proved by the combination of the two images in such a way that the parts that deviate to the nasal side constitute the nearer image; the parts that deviate to the temporal side, the further image. The stereoscopical effect is, however, more distinct and convincing with the coloured letters than with the strip used by Brücke.

The cause of the relative removal of the differently-coloured images lies in the excentricity of the pupil, as may be demonstrated experimentally. The pupils may be made highly excentric by covering them partially. Partial covering on the nasal side is equivalent to a removal of the pupil to the temporal side, and conversely, covering the temporal side is equivalent to removal to the nasal side. With a nasal excentric pupil a shifting of the differently-coloured images in one direction will be observed; with a temporal excentric pupil the shifting will be in the other direction.

The effect of an artificial excentricity of the pupil is surprising when both eyes are used. Anyone who sees the red

[1] Vorlesungen über Physiologie Wien, 1884 3 Aufl. B. 2, S. 95.

letters before the blue has only to cover his pupils symmetrically on the temporal side when he will observe that the red letters retreat and soon appear to be behind the blue. On covering his pupils symmetrically on the nasal side, the red letters come forward more and more, and seem at last (experimenting at a distance of four or five meters) to remain several decimeters in front of the blue. A person who sees the blue letters before the red has only to cover his pupils on the nasal side when he will observe that the distances change, the red coming forward and the blue shrinking back.

Lately, however, Dr. A. D. Waller has written to us that on repeating the experiment with a seemingly slight modification, he obtained the same effects with one eye alone. He used as a test object rings of blue paper on a red ground, or of red paper on a blue ground, and found that the nasal pupil of the left eye gives the same appearance of circular trenches or hillocks as does the temporal pupil of the right eye.

This observation has been the motive to a more thorough study of the phenomenon.

We shall in the first place try to describe the phenomenon exactly, mentioning all such conditions as tend to give it distinctness.

On looking with the right eye and a temporal pupil at red rings on a blue paper, the rings appear as circular hillocks when the paper is held to the left, and also sloping in that direction. One seems to be looking against the dark edges of a thick red ring fixed upon the blue paper. With a nasal pupil the red rings appear as circular trenches.

The phenomenon is the more striking in proportion to the purity of the colours used. The pupil must be made sufficiently excentric and in a suitable direction by means of a black screen that covers it from one side, or better still, by means of a stenopæic apparatus. The pupil must not be too narrow, and the whole eye should be wide open and

well directed, so as to avoid any partial covering by the nose, eye-lid or lashes. Lastly, we would point out that it is not desirable to keep the excentricity of the pupil constant for more than a brief period. For if one stares at the rings a long time with unmoved pupils, all appreciation of distance is lost, as in so many cases where only one eye is used, and the rings may even seem to lie in a plane that intersects the plane of the blue paper perpendicularly. If, on the contrary, one shifts the screen or the stenopæic apparatus now and then, the rings appear to rise and sink, and, under the above-mentioned conditions the rising will be with temporal pupil, the sinking with nasal pupil, and in a way almost as striking as if they were seen stereoscopically.

Fig. 2.

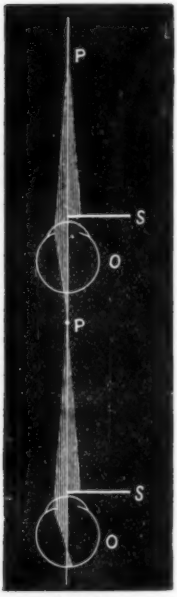


Fig. 1.

The explanation of the phenomenon will be found in the appearance of shadows.

If one looks with one eye and excentric pupil at a red and a blue light, one sees the red shifted with respect to the blue, in consequence of the chromatic aberration of the eye. The direction of displacement depends upon the direction of excentricity of the pupil, as is represented by figs. 1 and 2. In fig. 1 S represents a screen which covers the half of the pupil of the eye, and P is a blue light. In fig. 2 S has the same signification, but P is a red light. On the retina red is removed to the opposite, blue to the same side of the screen, so red must be projected outwards to its same side, blue to its opposite side. On looking with one eye at blue and red stripes which are adjacent to one another (see fig. 3) red will be seen removed to A, blue to B, when the pupil is covered from the side of A. Near m, the stripes will overlap one another, near n and l they will be shifted away from

one another. A purple border therefore appears near *m*, having an intensity of light equal to the sum of the intensities of a red and a blue stripe, whilst near *n* and *l* dark borders will be seen.

If the screen covers the pupil from the side of *B*, *l* and *n* will be light and *m* dark.

What has been said of the coloured stripes also holds good, of course, of the red rings on a blue ground, or blue rings on a red ground; they show light and dark borders in the same way when looked at with one eye and an excentric pupil.

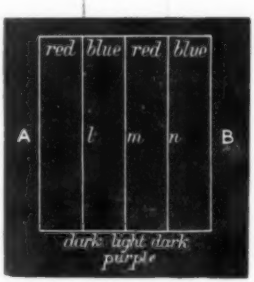


FIG 3.

Leaving for a moment the colours, we must now treat of the shadows that are seen when there is a small but steep depression or elevation on an equally lighted (uncoloured) and flat ground.

Our first enquiry is as to the apparent illumination of the flat ground and of the edges.

If the light falls in parallel rays and perpendicularly to the ground, the steep edges *a* and *b* (see figs. 4 and 5) will remain dark. If the light falls obliquely, they may receive either more or less illumination than the ground. If the light falls slopingly from the side of *A* (see fig. 4) or from the sides before or behind the plane of drawing, *a* will remain dark with all degrees of inclination of the rays. And even when the rays proceed from the fourth side *B*, *a* will receive less illumination than the ground, if the inclination of the

rays is more than 45°. Only in the case of the rays proceeding from B, and at a slope of 45°, or less, will a be as much or more highly lighted than the flat ground.

In the same way an edge, a or b, in fig. 5 will, in by far the greater number of cases, be darker than the ground.

FIG 4.

FIG 5.

We now proceed to give a mathematical treatment of the subject. In the midst M of a sphere (see fig. 6) imagine a flat ground with a small perpendicular edge, just as in fig. 7, and imagine the surface of the sphere infinitely large with respect to the surface of the ground. The latter lies in the horizontal large circle $ACBD$, the edge v v_1 (fig. 7) lies in the vertical large circle $CFDE$.

Let us suppose that the rays which fall from a single direction are reflected diffusely, so that the intensity of the reflected light, or the apparent illumination, depends only on the strength of the incident light and the angle of incidence.

Let us ask, in the first place, what chance there is of the flat ground being more highly lighted than the rising edge when the rays come from a single direction and all directions are equally probable?

Construct the large circle *CHDG*, which intersects the rectangular biangle *ECDB* in the middle, and from the point *P*, arbitrarily taken *below* that circle on the surface of the sphere, draw a ray *PM*, and then draw PP_1, a perpendicular on the plane *ACBD* and PP_2, a perpendicular on the plane *CFDE*. If *PM* represent the intensity of the incident light, PP_1 and PP_2 will represent the respective intensities with which the flat ground and the edge are lighted.

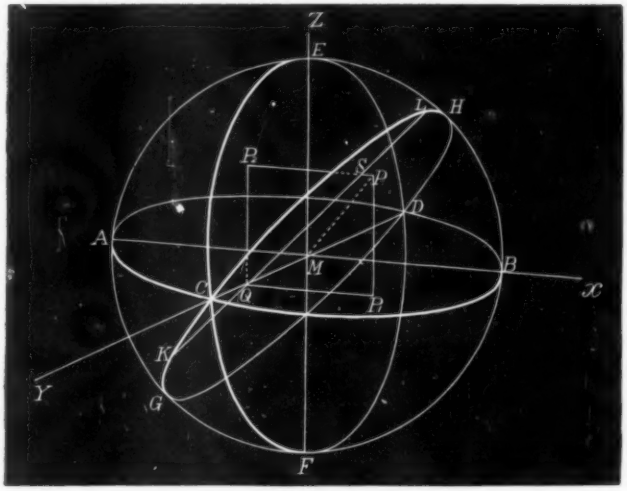


FIG 6.

Lay a plane through PP_1 and PP_2, this intersects the horizontal large circle *ACBD*, according to the line P_1Q, and the perpendicular large circle *CFDE*, according to the line P_2Q. PP_1QP_2 is a rectangle, which is intersected by plane *CGDH*, according to the line *QS*. The line *QS* cuts the angle P_2QP_1 in the middle. As the point *P* is taken *below* the plane *CGDH* on the surface of the sphere, the point of intersection *S* must lie between *P* and P_2, therefore PP_2 is longer than PP_1, *i.e.*, the edge receives more light

than the ground. If P were lying *in* the circle $CGDH$ on the surface of the sphere, the apparent illumination of both planes would be equal; if it were lying *above* that circle, the ground would receive more light than the edge. The chance of the latter being the case is expressed by the proportion of the curved surfaces of the spherical biangles $ACDH$ and $BCDH$ *i.e.*, $= 3 : 1$.

When we take into consideration that, in order to still be able to see the ground, the quantity of light which is reflected by it, cannot be diminished below a certain value, it is obvious, that P is to be taken at a certain distance from the horizontal circle $ACBD$, and this increases the chance of the ground being more highly lighted than the edge.

Let us ask in the second place as to the illumination of the

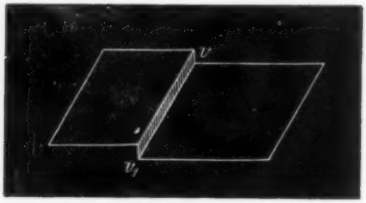


Fig 7.

horizontal and of the perpendicular plane, when the rays fall from all directions at the same time and with equal intensity. It is easily understood that in this case the horizontal ground will be lighted by the surface of half a sphere, whilst the perpendicular edge receives rays from only a quarter of the sphere-surface; the proportion of the apparent illumination will therefore be $= 2 : 1$. In reality the illumination of a ground will in most cases be a combination of stronger light from one side with weaker light from all other sides; therefore, the chance of the ground being more highly lighted than the edge—already greater than 3: 1., with illumination from a single side—will under ordinary circumstances be considerably increased. Generally, therefore, an observer looking at the steep edge of a depression or elevation on a flat

ground, will see this as a dark shadow. On the other hand, under certain circumstances, he will involuntarily conclude merely from the appearance of a dark border, that he is looking at a steep edge.

That this is the case, when under the above conditions, one looks at red rings on a blue ground or blue rings on a red ground, and sees them as hillocks or trenches, can easily be proved by looking at the rings, first from one side, and then from the other, keeping the pupil excentric in the same direction. Experimenting in the way described on p. 193, and looking at red rings on a sheet of blue paper with the right eye and temporal pupil, the red is apparently removed

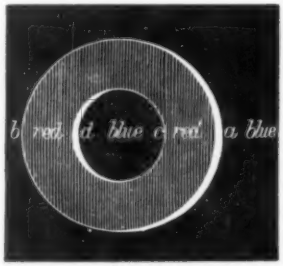


FIG 8.

to the left, the blue to the right, the ring showing a shadow on its outside at the right (fig. 8 *a*) and on its inside at the left border (*d*).

The sheet of paper was held to the left side, sloping in the same direction, and the red appeared to lie upon the blue. *On removing the sheet of paper to the right, and making it slope in that direction, the shadows will be formed with the same temporal pupil on the same places, but they are now looked at from another direction. Therefore, instead of rising above the blue, the red will appear to sink* (*figs.* 9 *and* 10).

If the sheet of paper is held on the same side sloping in the same direction, the left eye will see with nasal pupil what is seen by the right eye with temporal pupil; for the

pupil is not to be removed to the symmetrical, but to the same side, in order to shift the colours on the retina in the same direction

With pupil excentric in an upward or downward direction you have only to hold the coloured paper straight before you, and sloping forwards or backwards in order to cause the same

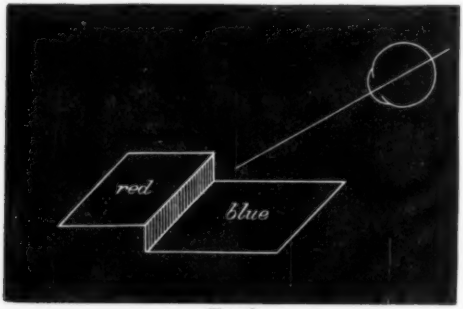


FIG 9.

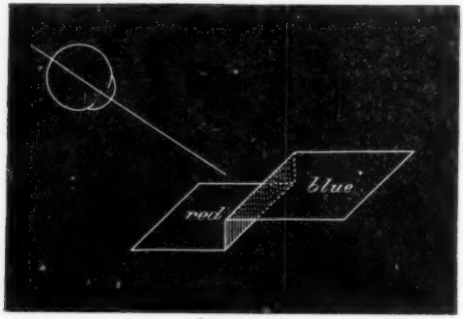


FIG 10

illusion; and the effect may be obtained equally well with the pupil excentric in all other directions, provided the sheet of paper be removed in the same direction or the opposite one, and that it turn on an axis which crosses that direction perpendicularly and lies in the plane of the paper.

As is mentioned on p. 195, purple-coloured light borders are observed on the rings near the dark borders. When they do not catch the eye too much, they seem to confirm the illusion by antithesis. On experimenting with a half-red, half-blue coloured paper, a single straight line of demarcation was shown, and red appeared before blue or blue before red, under the same conditions as with the coloured rings, when there appeared a dark border; when, however, a light border was seen no striking difference in distance could be noticed.

We have still, in conclusion, to treat of the question in how far the shadow that is thrown on the ground by a rising edge can affect the illusion mentioned. And here it must be

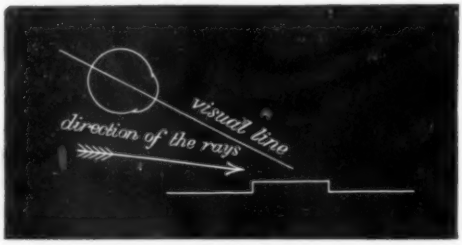


FIG 11.

taken into consideration that the light falls only in two directions, one opposite to the other, as when experimenting with the coloured rings we only see shadows lying either towards us or in a direction straight away from us.

If the light proceeds from the one side, the shadow that is thrown will enlarge the dark stripe of the rising edge, and thus strengthen its impression. If the light falls from the other side the shadow thrown will ordinarily be invisible to the eye of the observer.

An exception to this occurs with such position of the eye and direction of rays as is represented in fig. 11, where moreover the edge is more strongly lighted than the flat ground. We investigated the manner in which the coloured

rings were judged as hillocks or as trenches under these conditions. The experiment can easily be performed. We did it by means of direct sunlight, and found that the trenches, as well as the hillocks, were formed in the same manner as with the usual light.

We thus learn that notwithstanding that we know in a particular case the exact direction of the rays and the spot where, under these given conditions, the shadow *should be* formed, yet a visual impression is judged as if the shadows were caused by rays falling in the directions of most frequent occurrence.

ON ATTACKS OF LETHARGY AND ON HYPNOTIC SUGGESTION.[1]

BY PROFESSOR HITZIG.

Although, on the whole, I am not very fond of casuistics, I beg leave to call attention to a single case of hysteric sleep, which I have successfully treated by the employment of hypnotic suggestion. I do so, not only to submit this method of treatment for criticism, but because the case appeared to me to be not altogether without value on account of its ætiology, the peculiarity of its course, and on account of some observations which were made on the metabolism before, during, and after the sleeping fits.

A labourer, twenty years old, met with an accident in the night of the 11th of July, 1891, working a dredging machine. A bucket became loose and fell on his left arm, and a connecting-rod on the left side of his head. He sustained some considerable wounds of the skin and muscles of the extensor side of the left fore-arm, and some minor cuts on the left side of the head near the middle line. General symptoms were wanting; indeed, he was able to call upon the doctor himself.

He was further treated in a hospital, where the wounds on the head healed in four or five weeks, and those on the arm in about twelve weeks.

About the middle of August, according to the statements of a medical report, at intervals of from two to three weeks: and, according to the statements of the patient, at first every eleventh or twelfth day [subsequently every Tuesday or Wednesday], *states of excitement* began to set in, accompanied by headache and *rise of temperature*, upon which, as soon as the latter had reached 39 C, *a period of sleep* regularly followed. The sleep usually lasted from the evening of one day till noon of the third day, so that the patient slept nearly two days. The increase of temperature, which at the beginning of the attacks was said to reach 39.9,° become later less and less marked, until the temperature during sleep was stated to amount to only 36.3° C.

[1] Read before the Congress of Experimental Psychology, London, 1892.

During the sleep complete amnesia prevailed. During that time the patient never took any food, but during the attack he repeatedly drank, and discharged urine. The nurse, it was said, pressed, for this purpose upon the scar on the arm, whereupon the patient became awake for a few moments, and asked for a drink. Even this, however, he only learned from the lips of others.

After the attack regularly, but only for a short time, great faintness prevails, and a violent headache; after that the patient begins to feel very well, and to take abundant food.

Present State.

The patient, a middle-sized, strongly built and well-fed man, exhibits on the left parietal bone, about 3 cm. from the middle line, some movable scars, which are not painful when pressed. But another spot, of the size of a finger-joint, is painful when pressed, not so much when tapped; it is situated about the breadth of three fingers to the left of the sagittal suture behind the other scars, just midway between the coronal and lambdal sutures.

On the dorsal aspect of the left forearm there is a large red scar, forming an irregular acute angle; transversely it measures 8 cm., and reaches from the medial edge of the ulna obliquely upward to the middle of the radius, its longitudinal portion, 5.5 cm. long, lying above the radius. Across the middle of the fore-arm, corresponding to the scar and below it, there is a deep cavity caused by muscular defect; it deepens when the muscles are contracted, and at the same time the transverse portion of the scar is displaced with the muscles. On the dynamometer, the patient presses 55 kg. with the right hand, 21 kg. with the left.

When the face is not moving, the left naso-labial furrow is much more marked than the right one. When one-sided movement takes place, this difference is also clearly to be seen, and much more so when bilateral movement reaches its maximum. *Sensibility* is altered only near the scars, so that touches with a brush and pin-pricks are felt more painfully about a finger's breadth above them, and about 7 cm. laterally and below. Pressure on the transverse portion of the scar, and on a spot immediately below the middle of it is very painful, and it produces abnormal sensations in the back of the head. All this region is very sensitive to the touch of the shirt, and there is slight left-sided ovarian tenderness.

The knee-jerk is increased, and also produced by a blow on the tuberositas tibiæ.

Other anomalies are wanting.

When I saw the patient, for the first time, on the 15th of February, 1892, I told the assistant physician, with a suggestive intention: "You will see that the patient instantly falls asleep as soon as I look in his eyes." In fact, after I had fixed his eyes for a few seconds, I had put him into the hypnotic sleep. Afterwards hypnotism, easily produced by any means, was usually effected by the humming of a deep tuning-fork.

In his hypnotic state a complete left-sided hemianæsthesia was suggested to him for clinical purposes. Therefore, when I presented him in the *Clinique* on the 24th of February, he was completely anæsthetic all over the left side of his body to all sorts of stimulation, even the strongest induction currents. He had besides lost the sense of smell in his left nostril, and that of taste in the left side of his tongue. His organ of hearing had not been affected, but I suggested to him in a high degree a concentric restriction of his field of view in his left eye, and in not so high a degree in his right one. I succeeded in doing so, first by shrinking in a high degree both fields of vision, and then by widening the right one, as far as I pleased. So he was, on the whole, a specimen of a hemianæsthetic patient, as it is principally known to us by Charcot's classic descriptions.

After the demonstration, I made him fall asleep, and, by a short verbal suggestion, I made the whole hemianæsthesia disappear again before the clinical students.

The hypnotic state was made use of for some other ends besides. First of all, I thought it useful to render *anæsthetic* once for all, *the painful scars* on the arm of the patient. It is well-known since the publication by Pitres that painful spots on different parts of the body may play the part of so-called "points hypnogènes," which I need not explain here more in detail. Though simple stimulation of the scars did not produce any fit of sleep, still a relation between the scar and the fits is made probable, for the patient, who did not react against other stimulation, could be awakened for a short time by pressing the scar. So, for my part, I am of the opinion that scars are much more commonly causes of hysterical than of epileptic attacks. I, therefore, wished to exclude from the sensorium the painful stimulation, which was produced by irritating the scars. I placed my confidence in the effect of anæsthetizing by suggestion; but isolated anæsthetizing of the scars miscarrying, I was obliged to anæsthetize the whole fore-arm. The effect of such a suggestion done but once, lasted for several months; so far completely, that

the patient very soon did not perceive a touch at all, and painful stimulations he only felt as a touch. I do not think it impossible that the diminution and the temporary suppression of the prodromal loss of weight, of which I shall speak further on, is to be explained in the same way.

Hypnotic suggestion was next employed against prodromal headache, partly with complete, partly with, but passing success.

Lastly, I employed it against the fits of sleep. However, before discussing this application of the treatment, I shall describe the course of the fits of sleep themselves.

The patient had finished his last period of sleep out of the clinique, exactly eight days before the setting in of the first fit, which attacked him in the clinique. On the whole, he had seven fits in the clinique; each of them was preceded by a prodromal stage.

Usually, about two days before the setting in of the sleep, the weight of the body began to decrease. At the same time, pain and humming in the head began; the expression of the face, cheerful and open before, became distrustful and irritable; in a similar way, he was mentally changed, and the nearer the fit approached the more his eyes closed, so that one could judge from the patient's eyelids hanging down that a fit was approaching.

The fits of sleep, except the last one, which was stopped, lasted from 34 to 36 hours, once 41 hours. *The intervals* lasted from 7 days and 13 hours to 13 days and 13 hours. *During the fits* the patient usually reclined quietly on his back; sometimes, however, he tossed about. This principally took place after 15 cgrs., churrus (a very active species of cannabis indica) had been given to him during his fit. The eye-lids did not quiver. Contractions or convulsions, especially of the masseters, were not present. The eyeballs were directed upward, and when the lids were kept open for a time, slowly moved to and fro. His temperature, pulse, and respiration did not show any considerable changes; the last two only when churrus had been given.

The patient could be awakened by any means, sometimes, by pressing the left ovarian region or the scar; then he asked for water, drank and passed urine, whereupon he at once went to sleep again. Once, on the 2nd of March, he awoke spontaneously and behaved in the same way.

During his sleep, the weight of the body, the volume, the specific gravity and the quantity of nitrogen in the urine decreased. The secretion of phosphoric acid was not examined.

After his sleep, the patient had a more or less violent headache; soon, however, he began, with a good appetite, to eat freely; the weight of his body rapidly increased; in the same way, the volume, the specific gravity, and the nitrogen of the urine increased. The patient then felt very well, worked hard indoors and in the garden, and betrayed no signs of a hysterical character till the prodromata of the new fit began to act.

I said above that the patient once had a long interval of 13 days between his fits. It lasted from the 21st of March till the 3rd of April. It is to be noticed that during this time, *i.e.*, on the 25th of March his usual prodromata took place, and on the 27th of March very free bleeding of the nose occurred. Then he felt as if he had gone through his sleep, and, indeed, between the bleeding of the nose and the sleep, there was the same mean interval as usually took place between two sleeping periods. In this case, evidently, the bleeding which interrupted the regular course of the symptoms took place as an equivalent of the fit. Then I tried suggestively to interrupt this course in different ways, supposing that if this end could be really attained, the fits would be effectually stopped. For this purpose, I hypnotically suggested to the patient several times in the interval that the fits would leave between them a pause longer than usual and would not reappear before a certain day appointed by me. Then I suggested to him during the sleeping fit that the fit would last a shorter time than usual, in fact, till an hour appointed by me. These experiments, however, had no result whatever.

Then I hypnotized the patient on the 26th of April, in the afternoon, during the prodromal stage, when he had a violent headache, and when from his half-closed eyes it might be with certainty supposed that his lethargic sleep would occur that night. Then I suggested to him that I had just put him into a magnetic sleep, that this sleep never passes into his morbid sleep, and that though I had not been able to awake him from his morbid sleep, I should awake him from his present sleep. This would take place on the next day. I should three times clap my hands, the third time he would awake, but not before. In this way he would be cured; he would never again suffer a new fit.

I adopted this process from reasoning that, since experience shows that the effect of suggestion usually (as was, in fact, the case of our patient), decreases on account of other sensations intervening, suggestion ought to have an extraordinary effect, if it were made as near as possible to the impending fit, if it were applied during the hypnotic state, and if by inducing the hypnotic

state external stimuli were excluded, as much as possible during the interval. As a matter of fact the result agreed with this reasoning.

On the 27th of April, 22 hours after the setting in of the hypnotic state, the patient awoke when I clapped my hand for the third time. Since then, that is, during more than three months, he has never had a fit. Nevertheless, the patient is by no means completely cured. His prodromal symptoms described above still occur, but they appear, it is true, in irregular and longer intervals, of from a fortnight to three weeks, and his head-ache is not so intense as before.

These symptoms still usually last two days, as before, and they are accompanied with the former irritability and excitability of his state of mind. The patient says that though he remembers all the events of those days, yet he afterwards fancies that he did everything in a dream. Besides this, the scar and its neighbourhood always becomes sensitive, as soon as I remove the anæsthesia of the fore-arm.

In this case we again draw the conclusion that, by suggestion, we can remove some hysterical symptoms, but that by it alone we are not able to remove the hysteria itself. The patient has been submitted to a therapeutic treatment, but it has so far not proved completely successful.

Now, I desire to call attention to some points of very particular interest. First, as regards the character of these pathological states of sleep, I am of opinion that, from the recent investigation by Charcot and his school, above all by Gilles de la Tourette and Cathelineau, by Lowenfeld and others, there can be no doubt but that they belong to hysteria. Therefore, I do not hesitate to include the fits of our patient among hysteria, though, in this case, not only all the symptoms of hystero-epilepsy were wanting, but also all the other symptoms of hysteria, except the ovarian, the susceptibility of being hypnotized and the possibility of suggestion. From this very reason the case seems to me to claim a greater interest, because differing from the majority of the cases hitherto published. It gives a very clear idea of lethargy occurring in a marked degree, and modified only by our various operations upon it. Further, agreeing as I do with the doctrine of Charcot and his followers, I am inclined to consider the sleeping fit as a part of the hystero-epileptic attack, and in our case, therefore, as an abortive fit. Not only the general impression, the prodromata, the fit and the post-hysteric state strikingly remind us of the analogous states of epi-

lepsy and hysteria, but, in our case, there is also a certain, though not complete, confirmation in the very peculiar changes of nutrition with those in the fits of *grande hystérie*, which according to the investigations by Gilles de la Tourette and Cathelineau, are identical in the various other phases of the hystero-epileptic fit and the sleeping fits.

In the second place, the ætiology of the disease is full of interest. Without any doubt we have to do here with a functional neurosis of hysteric character caused by a trauma. It is not in the least my intention to enter into the dispute on traumatic neurosis which of late has been so vigorously conducted. Suffice it to say that I have seen a very considerable number of traumatic diseases of the central nervous system of a functional character, and that they may be divided without any difficulty into hypochondriacal, neurasthenic and hysterical forms. The latter, though not so common as the other forms, are not at all rare, considered by themselves. Therefore, I do not regard with approval the view of certain authors who, because they have not observed such cases, or because they did not recognise them, declare the occurrence of hysteria and hypochondria in persons of low extraction, above all in males, and especially after trauma, to be exceptional, and who assert that other authors, who in this respect command a greater experience, have been deceived by dissemblers. The nutritive processes before, during, and after the sleeping periods, seem to me particularly worthy of the attention of scientific men, because their study may be presumed to afford a nearer insight into the changes of the cerebral mechanism occurring in the fits of *grande hystérie*.

The few facts I can impart in this respect differ in some points from the observations of Gilles de la Tourette and Cathelineau. These authors have stated that the nutritive troubles described by them did not begin before the fits set in, and ceased with them. This did not apply in my case. Before and during the first two fits, the patient lost 4 and 3 klg. respectively. I cannot tell how much of the loss is to be put to the account of the prodromal period, because daily weighing was not then made. During the prodromata of the three following fits, however, he lost 2 kg., 750 g., 2 kg. and 1 kgr. Before the sixth fit no loss of weight was to be detected, and before the seventh fit, which was the last, it amounted only to 500 gr.

You will have noticed that these figures, except the last, show a continual decrease, and I once more insist upon the

possibility of the improvement being caused by the diminution of the prodromal headache, perhaps as well by suggestive anæsthetisation of the scar. There is, however, an inverse ratio between the decrease of the weight of the body during the fit and the decrease before the fit; the latter more and more diminishing, the former augmented in the same degree, so that the addition of these two figures always yielded the same weight during the whole of a fit, that is twice 3 kgr. 500 gr. and twice 3 kgr. 250 gr.

Not only the weight of the body, but also the excretion of nitrogen showed considerable changes during the prodromata.

In order to be surer of my ground, I desired Professor von Mering to ascertain the nitrogen, which he did according to Kjeldahl's method. Unfortunately, the results furnished me did not form a complete series, so that I must confine myself to stating them simply. Thus, the secretion of urea increased from 24.6 gr. on the day before the setting in of the prodromata of the fourth fit to 30.21 gr. during the first day of the prodroma, and to 44 gr. during the second. The patient having headache on these days, and therefore certainly not having consumed more food than usual, but having lost considerably in weight, the augmented excretion of urea may with certainty be put to the account of the loss of weight.

Easier to explain is the augmented excretion of nitrogen immediately after the fit, which, in three fits, reached its highest point with 37.95, 33.25, and 30.4 gr. of urea, on the day after the fit, for it has been ascertained that on these days the patient consumed a much larger quantity of food. During his sleep, as has been stated, the secretion of urea diminished, corresponding to the statements of Gilles de la Tourette and Cathelineau. The lowest standard noticed during a complete sleeping day amounted to 5.93 gr.; the patient, at the same time, ate nothing, and sustained a considerable loss of weight. The latter, therefore, may be put (or to a large extent) to the account of the metabolism destroying tissue. The volume of urine generally fluctuated very much, but during the five fits in which it could be measured, it showed, relatively speaking, great regularity. It was diminished, and reduced on the basis of 24 hours, amounted to 1000, 987, 770, 867 and 800 ccm. In the last fit part of the urine was lost.

Of course there was, during the intervals between the fits, an inverse ratio between the specific gravity and the volume of the urine, and it was, therefore, also very fluctuating. But on the complete sleeping days it was very constant, that is, 1017, 1018,

1016 and 1017. On the sleeping day, which had been ushered in by the hypnotic sleep, it amounted to 1020.

I am very far from drawing far-reaching inferences from the few observations which I have given above. I think, however, I am justified in saying that the question of the nutrition during the hysterical sleep is not yet answered by the formula.

"Little metabolism occurs in hysterical persons during the fit": this formula contains only a part of the whole truth. Finally, since many command a much more extensive store of observations on hysterical persons than myself, I beg to call attention to the mode of cure which I applied. I am far from thinking that persons with inveterate hystero-epileptic fits can be cured by anæsthetizing hysterogenic zones, or by stopping their sleeping fits by means of hypnotising and suggesting at the same time. But since every outbreak of inveterate fits is preceded by a period more favourable to our therapeutic operations, the experiment of leading the disease into another channel during this period, in the manner I have described, seems to me worthy of trial.

Month	SLEEP.		INTERVAL.		Loss of weight during sleep.	Loss of weight before sleep.	Increase of weight after sleep.
	Days.	Duration.	Days.	Duration.			
February.	9th ev. to 11th noon.	40 hours.					
February.	19th 2 p.m. to 21st 8 a.m.	42 hours.	11th noon to 19th 2 p.m.	8 days.		4 kgr.	5.5 kgr.
March.	1st 3 p.m. to 3rd 7½ a.m.	41 hours.	21st Feb. 8 a.m. to 1st March 3 p.m.	9 days 7 hours.		3 kgr.	3.5 kgr.
March.	10th ev. to 12th 8 a.m.	34 hours.	3rd 7½ a.m. to 10th ev.	7 days 14 hours.	0.750 kgr.	2.750 kgr.	5.5 kgr.
March.	19th 9 p.m. to 21st 8 a.m.	35 hours.	12th 8 a.m. to 19th 9 p.m.	7 days 13 hours.	1.5 kgr.	2.0 kgr.	6.0 kgr.
April.	3rd 9 p.m. to 5th 8 a.m.	35 hours.	21st 8 a.m. to 3rd 9 p.m.	13 days 13 hours.	2.250 kgr.	1.0 kgr.	2.5 kgr.
April.	13th 9 p.m. to 15th 9 a.m.	36 hours.	5th 8 a.m. to 13th 9 p.m.	8 days 13 hours.	3.250 kgr.		3.0 kgr.
April.	26th noon.		15th 9 a.m. to 26th.	11 days.	2.0 kgr.	0.5 kgr.	3.750 kgr.

ON DIFFUSE ENCEPHALITIS: WITH AN ACCOUNT OF A CASE[1] IN WHICH THE PATIENT SURVIVED.

BY R. LAWFORD KNAGGS, M.C.
AND
R. CONYNGHAM BROWN, M.B.

JAMES C——, age 10, was admitted into the Huddersfield Infirmary, on October 3, 1891, under the care of Mr. Samuel Knaggs, for spastic paralysis of the left arm and leg and suffering from fits.

He was the son of healthy parents, and one of a healthy family.

He had perfect health till five years before, when he fell from a wall seven or eight feet high. He was taken indoors, and, though apparently none the worse for the fall (?) it was thought advisable to put him to bed. The following morning he could not be roused, and a medical man was sent for, who "said he was suffering from compression." The greater part of that day he lay unconscious, and then gradually recovered; and in a few days was going about as usual. A month later, without any apparent cause, he fell down in a fit; he was unconscious, and "twitched his left arm and leg." After an interval of two months he had a similar fit, and, for a period of two years, the fits recurred every other month. Between them the boy appeared all right, but for slight loss of power on his left side, which was noticed towards the close of that time.

Nearly three years before admission he had a second fall; and though it was only from a height of two or three feet he was taken up insensible. For two weeks he lay unconscious and perfectly quiet, the only change being slowly increasing emaciation; and then, though gradually regaining strength, he was unable to rise from his bed for eight months.

[1] The writers are indebted to the kindness of Mr. Samuel Knaggs for permission to publish this case, and to Dr. F. L. Mackenzie for some valuable notes about the early history of the illness.

Dr. F. L. Mackenzie, who was called in to see the patient somewhere about this time, has kindly supplied us with the following notes of his condition. On January 9, 1889, he saw the patient for the first time, and "found him in an extremely weak and emaciated condition. He lay on his right side, his eyes half open, and without any other characteristic facial condition. He was coiled up, his head being flexed upon his chest, and both thighs acutely flexed on the abdomen. When shouted at, or shaken somewhat roughly, he responded with a howl and again became quiescent. There was no evidence of hemiplegia. The emaciation was so extreme that unilateral wasting could hardly have been detected. His pupils were equal, and only slightly responded to light. The conjunctival reflex was very imperfect, and so was the knee tendon reflex." "Urine and fæces were passed in bed. Respiration was regular, and not indicative of cerebral disease. P. 120, small. Temperature 97.8." "In the course of a week the patient began to move in bed, and take more nourishment."[1] By the middle of April, his general condition was greatly improved, and he was moderately intelligent, but "as movement of the limbs returned, evidence of hemiplegia showed itself—flattening of the left lip, slight contracture of the left hand, and flexion of the left leg" (which last was corrected by extension). The condition of spastic paralysis, which the arm presented on admission, gradually developed, the flexion being first noted at the wrist, and then at the elbow.

During the eight months the patient was in bed, he had no fits, but shortly after getting up he had one, in which he is stated to have squinted and worked the left side. These fits continued with increasing frequency till his admission. Dr. Mackenzie was again consulted on September 7, 1889, and describes "attacks of sudden jerking, especially of the left leg"—"a sudden clonic spasm sometimes causing the patient to fall." "There was no loss of consciousness, no aura and no drowsiness after the attack." At first, these attacks occurred two or three times a week, but they practically disappeared under treatment by the end of November. In March, 1891, Dr. Mackenzie saw the boy again for "similar attacks affecting the upper extremity more markedly than formerly." They occurred now as often as two or three times a day, and treatment had no effect upon them.

With the increase in number of the fits, the loss of power on the left side became more marked, and for 18 months previous to

[1] Mercury was given during this period, and its value seems to have been very marked.

admission his parents stated that he had become peculiar in manner and difficult to control.

On admission (October 3, 1891) the patient was a powerfully-made well-nourished, healthy-looking boy. His bright eyes gave him an intelligent expression, but his slowness in answering questions, deliberate enunciation, and eccentric behaviour evidenced an impaired intellect.

His gait was peculiar and jerky, owing to the spastic condition of the left leg, which was advanced without bending the knee. The jerkiness was communicated to the left arm.

There was slight left facial paresis and a spastic paralytic condition of the left arm and left leg.

The left arm was always held close into the side. The hand was bent at right angles to the fore-arm, and the phalangeal joints were flexed. The fore-arm was pronated and bent almost to a right angle with the arm.

The hand was smaller than the other, and the limb slightly thinner than the right.

The deltoid was particularly flabby. The arm could be abducted nearly to the horizontal by rotation of the scapula, but the deltoid was inactive. With effort he could partially extend the elbow, but, once extended, it was flexed without difficulty. There was hardly any power of movement in the wrist and finger-joints. There was considerable rigidity to passive movement, which was most marked in extension.

The left leg was extended. It was of equal length, but slightly thinner than the right, and the power of voluntary movement was only slightly inferior. The greatest difficulty was shown in extending the foot.

Rigidity to passive movement was present in flexion and extension, and especially in abduction.

On the left side there was slight ankle clonus and an exaggerated knee jerk. Sensation was normal on both sides. The fundus in both eyes was healthy, and no evidence of an old papillitis could be detected.

The heart, lungs, and abdomen were normal, and the urine had a sp. gr. 1020, and contained no albumen.

The boy was kept under observation for a fortnight, during which time he had at least three or four fits every day. They were of an epileptiform character, and showed a distinct tendency to become more frequent.

He had a stiff feeling in his left arm when they were coming on. During the fit he became unconscious and fell. The mouth

was drawn to the left side, and a stiffening of the left arm and leg was succeeded by clonic spasms; these, though most marked on the left side, were never entirely limited to it. A fit lasted about two minutes, and on recovering he became pale and faint for a few seconds and then said he felt well again.

The convulsive movements did not begin in any particular muscle or group of muscles. At other times the left arm would be involuntarily jerked forward once. This was of frequent occurrence and was unaccompanied by any other symptom. The temperature was normal, but when taken in the left axilla it usually registered nearly a degree lower (subnormal) than when taken in the right.

The history, and the unilateral and progressive character of the paralysis and of the convulsions pointed to some lesion of a traumatic character implicating the R. motor area, and after consultation it was decided to explore over that part of the brain.

Operation. On October 16 (the boy's head having been previously shaved and rendered aseptic), the situation of the fissure of Rolando was marked out on the right side of the scalp, a large semi-lunar flap reflected down, and two half-inch discs of bone removed above and below a point situated over the arm centre. The dura mater did not pulsate very evidently at this stage, and it did not bulge. A third disc in front of, and intermediate between the other two was removed, the intervening bone marked out by Hey's saw, and clipped away with bone forceps, and in this way a triangular aperture was made whose sides measured from 2 to $2\frac{1}{2}$ inches. The exposed dura concealed the lower $\frac{2}{3}$ of the fissure of Rolando, and the centres for the face and arm, and part of the leg centre.

The dura did not bulge at all into the opening, and there was no pulsation, but the child's condition had rapidly failed and the absence of pulsation was thought to be connected with that fact. The flap was re-adjusted and further exploration postponed.

There had been a good deal of hæmorrhage from the flap and the bone, but not to such an extent as to raise alarm.

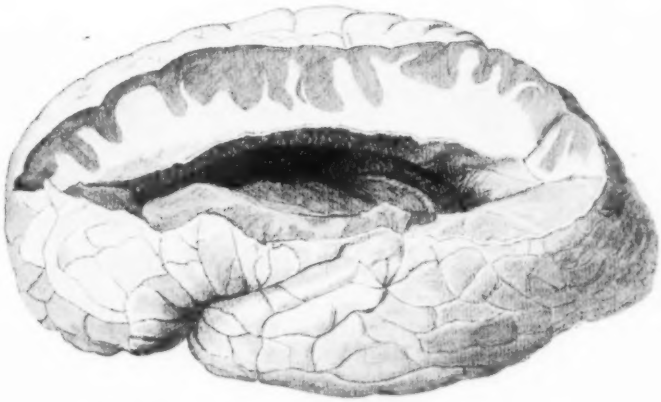
Before the operation $\frac{1}{6}$ of a grain of morphia was to have been injected, but owing to a defective syringe less than that quantity was probably administered.

At the beginning of the operation the child's condition was good, but no chloroform was given after the removal of the second disc.

When Hey's saw was being used the anæsthetist found it necessary to inject ether, and though the operation was concluded

DRAWING OF THE RIGHT LATERAL VENTRICLE.

(Case of J. C.) [*R. C. Brown, fecit.*]



DRAWING OF THE LEFT LATERAL VENTRICLE.

(Case of J. C.) [*R. C. Brown, fecit.*]

with all despatch and means adopted to lessen shock, the pulse could not not be felt at its conclusion, and the patient's condition was very serious. He was placed in bed with the head and shoulders depressed, and for twenty minutes or half-an-hour respiration had to be kept going by slight artificial aid. Then gentle breathing became established, and it was just possible to detect the pulse. During the operation, and till death occurred, some four hours later, the pupils remained contracted and very small.

Post-mortem.—The brain only was examined. The dura mater was quite normal, and there was no opacity or thickening of the arachnoid. There was œdema of the pia mater on the convexity of the R. hemisphere over the frontal and parietal lobes. This, no doubt, was due to the operation. The surface of the R. hemisphere was fluctuant and tremulous from within an inch of the anterior extremity of the frontal lobe as far back as the occipital fissure, and laterally from half an inch from the longitudinal fissure to the sylvian fissure. The same condition was marked in the middle temporo-sphenoidal convolution. It was plain that the R. hemisphere contained a cavity full of fluid, and that its walls were very thin. Eight ounces of fluid were removed with an aspirator, and the brain was placed in a bichromate preservative solution.

The fluid was clear, of an alkaline reaction, and had a sp. gravity of 1010. It contained a trace of albumen and sugar, and a considerable quantity of salt. Microscopical examination gave no information.

Examination of the hardened brain.—The R. hemisphere was decidedly smaller than the left. This difference was not noticed at the necropsy, and was probably due to the preparation of the brain and the removal of some of the fluid contents.

In order to expose the R. ventricle, its roof was removed by a vertical incision 6½in. long parallel to the median fissure and half an inch external to it. The extremities of this incision were joined by a horizontal cut which divided the sylvian fissure near its bifurcation. The roof so removed was 6½in. long by 3in. wide at its broadest part.

The dimensions of the R. ventricle were:

Length	4⅝in.
Breadth in ant. cornu	1⅛in.
,, in centre of ventricle	1 1/16in.
,, behind descending cornu ...	1¾in.

Depth, everywhere about 1¾in., except at the anterior part, where it was 2⅛in.

In shape it resembled the hollow cast of a large kidney, the dilated anterior cornu corresponding to the upper extremity, the posterior and part of the descending cornu, dilated into a single hollow, represented the lower extremity, whilst the concavity of the hilus was suggested by the rounded elevation formed by the corpus striatum and optic thalamus.

The *thickness* of the wall of the ventricle measured less than $\frac{3}{16}$ of an inch anteriorly, $\frac{1}{2}$ an inch posteriorly, and $\frac{14}{16}$ of an inch externally at its thickest part, where the section had traversed the temporo-sphenoidal lobe.

The anterior and posterior cornua were dilated beyond recognition, and formed part of the general kidney-shaped cavity. The descending cornu was enormously enlarged, and when the little finger was introduced to the end, its tip could be moved about as if inside the finger of a much too large thick glove.

The lining membrane of the ventricle was thick and hard, and could be partially peeled away here and there; the thickening was common to both ventricles, but the readiness with which it could be separated in the right as compared with the left was due to the firmness of the brain tissue surrounding the right ventricle.

The corpus striatum was so compressed and atrophied that its position could only be guessed at. The optic thalamus was visible in front of the choroidal plexus as an eminence $\frac{1}{2}$in. long by $\frac{3}{16}$in. wide. In front of this eminence was the foramen of Munro—an irregular opening having a diameter of $\frac{5}{16}$ of an inch. Reaching from this foramen and curving into the descending cornu was the choroidal plexus. The septum lucidum was thinned and expanded, and anteriorly two rounded apertures with smooth edges (diameter about $\frac{3}{16}$in.), and a third minute one communicated with the left ventricle. They were situated just above the foramen of Munro. The walls of the right ventricle looked, and were firm, and when the roof was removed the ventricle maintained its shape and showed no tendency to collapse. The part removed could be handled and raised by one end without any fear of damage resulting. This formed a striking contrast to the soft and succulent condition of the left hemisphere, which resembled fresh brain substance, although it had been in hardening fluids for some months.

The left ventricle was very different.—The roof was removed as on the right side, but on account of the thickness and softness of the walls it was difficult, and when it was lifted off precautions had to be taken to prevent the sides of the ventricle falling together.

The extreme length of the L. ventricle was ... 4in.
The width ,, ,, ,, about ... 1in.
The depth ,, ,, ,, about ... ½in.

These measurements cannot be considered exact, because, in consequence of the softness of the walls and the necessity for handling the parts in order to inspect them, the cavity could be made to appear either larger or smaller, but when care was taken to keep the structures as far as possible in their natural position, the cavity seemed to be approximately of the above dimensions.

The walls were nowhere less than an inch thick, and in some places as much as 1½inches.

During manipulation there was a tendency for the lining membrane with a layer of brain substance nearly ⅛in. thick, to separate from the surrounding tissue.

The anterior, posterior, and descending cornua were larger than natural, but the last-named would not admit the little finger.

The corpus striatum and optic thalamus were normal in shape and appearance; the foramen of Munro was enlarged, but smaller than that on the R. side, and had a more regular outline. The L. ventricle appeared uniformly and moderately enlarged, but there was no obvious atrophy of the surrounding structures.

Base of the brain.—On looking at the base the difference in size of the two hemispheres was very marked. The measurements were—

R. 6in. × 2½in.
L. 7in. × 3in.

The floor of the third ventricle formed a prominence as big as half a small walnut, and was about the thickness of a grape skin. It was divided by a median incision.

The third ventricle was dilated, and admitted the first phalanx of the index finger without risk of doing any damage. The R. wall was markedly concave—the left was flat.

Section through the pons showed the iter was not dilated. The tissue around it was slightly softened (*post-mortem* change?). There was no dilatation of, or atrophy of the structures in the *fourth ventricle*, and the cerebellum was unaltered.

The medulla showed descending degeneration in the usual places.

We are indebted to Professor Jacob for the following description of two microscopical sections cut from corresponding positions in the R. and L. frontal lobes.

Right.—"Considerably thinned. Microscopically has lost all trace of nerve structure, showing no nerve cells, but merely condensed neuroglia infiltrated with inflammatory products. Numerous inflammatory foci are present, and the perivascular spaces are enlarged.

"Pia mater shows marked inflammatory thickening."

Left.—"The ordinary layers of the cortex can be recognised, but the nerve cells show considerable degenerative changes, and there is much dilatation of the perivascular spaces. There is no thickening of the pia mater."

There were peculiarities in the clinical history of this case, which all who saw it during life were unable to explain upon any supposition. The pathological conditions throw light upon those clinical difficulties, and in the following explanation we shall try to show how the clinical facts and the *post-mortem* appearances can be interpreted.

The first injury occurred five years ago and, after a considerable interval of consciousness, was followed by slight signs of compression which soon disappeared. That some serious lesion had resulted was clear, for a month later a fit occurred beginning in the L. arm, and this was repeated every second month.

Now this is hardly like a history of hæmorrhage, and nothing was found at the necropsy to give colour to such a suggestion. But it is not at all improbable, in the light of subsequent events, that there may have occurred at this time a transient congestion of part of the encephalon with effusion, capable of producing slight and temporary symptoms of compression; and a bruised brain, which we might expect to be the initial factor in such a traumatic congestion, would easily explain the subsequent development of epileptiform attacks.

After an interval of two years another fall occurred. There is nothing to show whether this was the result of a fit or of accident. Concussion resulted, and from the prolonged unconsciousness (about a fortnight) that ensued it is practically certain that the patient was again suffering from compression. During a very slow recovery he exhibited for a time symptoms of cerebral irritation, and signs of spastic paralysis of the L. arm and leg gradually appeared.

He was unable to leave his bed for eight months, and the medical man who saw the patient at this time has described him as having a "purely vegetative existence."

The pathological appearances suggest that there occurred during this period:

(1) A sub-acute encephalitis involving the whole or greater part of the R. hemisphere, and producing a softening of the affected area.

(2) Inflammatory effusion into the brain substance, into the ventricles, and into the lymph spaces on the surface of the brain, giving rise to increased intra-cranial pressure and to signs of compression.

(3) Dilatation of the R. ventricle with absorption of the softened and inflamed brain tissue surrounding it in consequence of the increased intra-ventricular pressure; and

(4). From the same cause, dilatation of the L. ventricle, but without absorption, because the L. hemisphere was not involved in the inflammation, and was of normal consistency.

(5) Loss of function of those parts of the brain which had been or were inflamed.

Prolonged increased pressure would lead to absorption and recession of the softened inflamed brain tissue wherever it was exposed to it, whether upon the surface of the brain or within the ventricles, and since the ventricles are in direct communication with the lymph spaces upon the surface of the brain, any increase of pressure within the ventricles would speedily equalize itself throughout the whole cerebro-spinal canal.

In order, therefore, to explain why absorption of brain tissue has occurred only at the expense of the softened parts of the ventricle walls *from within*, it is necessary to account for the intra-ventricular contents being maintained at a higher tension than normal, whilst the pressure upon the surface of the brain hardly varied from what was natural; and this can be done if a plausible reason can be suggested to show that the intra-ventricular fluid was prevented escaping by the iter.

The maintenance of an ordinary pressure upon the

surface of the brain is easily understood, for any temporary increase of fluid resulting from inflammatory effusion would speedily be drained away into the general circulation.

Now when the R. hemisphere was inflamed the adjacent and continuous structures could hardly have been so fortunate as to escape entirely without feeling some effect from the mischief, even supposing that they were not directly involved; and it is not difficult to understand how so small a channel as the iter might be occluded by the pressure of inflammatory effusion or œdema in its surrounding tissue. And a further possibility may be mentioned—the orifice of the iter in the third ventricle may have been obstructed by an œdematous ependyma. In one or both of these ways in all probability the excess of fluid secreted into the ventricles was prevented escaping, the intra-ventricular pressure was raised, dilatation of both lateral and the third ventricles took place, and on the R. side the dilatation was not only more marked because this hemisphere was inflamed and soft, but was materially assisted by absorption from pressure of the damaged brain tissue, and at a later period doubtless by the contraction of the organized products of inflammation. When the encephalitis subsided the effusion into the cerebral tissues would be absorbed, the iter once more become patent, and the normal equilibrium of the cerbro-spinal fluid be restored.

If we consider how much of the substance of the R. hemisphere must have been destroyed at this period, partly from pressure and partly from the contraction of cicatricial tissue resulting from inflammatory exudation, there is no difficulty in accounting for the gradual onset of spastic paralysis in the L. arm and leg, which developed during the eight months the patient was confined to bed.

But what is the meaning of the peculiar condition of apathy in which the patient lay during the eight months following the period of compression?

It is probable that after the first two months the brain was in very much the same state as that in which it was found at the *post-mortem*, and it may be that the cause of this condition is to be sought in the loss or diminution of function that most structures are subject to during and after

inflammation. This would explain not only the tedious and very gradual recovery, but also the recurrence of fits which took place soon after he was sufficiently improved to be able to get out of bed, and which had been in abeyance from the time he met with his second fall. As the period of impaired functional activity gradually passed away the power to respond to irritation would reappear in those ganglionic and conducting structures that had not been too seriously damaged to recover.

Lastly, it must not be forgotten that at the time of the operation, and no doubt for months and even years before, the condition of parts was stationary so far as the macroscopic lesions were concerned;—one ventricle was enormously and the other moderately dilated; both were filled with normal cerebro-spinal fluid at the ordinary tension and free from all inflammatory elements. The conditions present were the result of a long past inflammation. The cerebro-spinal fluid was fulfilling its function of properly supplying the vacuum within the cerebro-spinal canal, and it was present in such large quantities because the potential vacuum had been so greatly increased.[1]

In attempting to discuss diffuse inflammation of the brain it is very difficult to find firm ground from which to make a start.

The microscopic appearances of chronic inflammation and sclerosis are apparently indistinguishable, and in doubtful cases the decision between inflammatory or purely degenerative changes can hardly fail to be arbitrary; and to those who are not experts in the pathology of the nervous system the indifferent application of terms, implying different processes of disease, to a particular morbid condition, is very puzzling.

The obscurity surrounding diffuse encephalitis is apparent in the account of inflammation of the brain given by Mr. Jonathan Hutchinson in "Quain's Dictionary of Medicine," vol. i., p. 148 (1883).

[1] The authors trust that the full description they have given of a case to which they have been unable to find a parallel may not be thought unnecessary. Full of interest to the student of medicine, it is most instructive as an illustration of one of the pitfalls that may be encountered in Cerebral Surgery.

"It may, perhaps, be doubted whether the occurrence of diffuse inflammation of the brain substance as an acute disease has as yet been proved, excepting as a result of wounds. Even as a traumatic lesion, its special features have by no means been accurately studied. It is, however, highly probable that after penetrating wounds of the brain, its substance may inflame, just as the cellular tissue of a limb may, the inflammatory processes beginning at the site of the wound, and rapidly spreading through a large part of the hemisphere. It is probably in the perivascular spaces that the process chiefly spreads, and it is in these that the microscope will detect the most abundant results. Such a condition of diffuse encephalitis may exist without there being any visible changes in the brain. It may, perhaps, be a little softened or a little congested, but very probably there is nothing about which the most experienced pathologist could feel certain until the microscope is resorted to."

"It is not possible, in the present state of our knowledge, to speak with any certainty of the symptoms of diffuse encephalitis. They will vary, of course, with the region affected; and disturbance of function, followed by, more or less, complete loss, will be the most frequent occurrences."

In the hope of learning something more definite about diffuse inflammation of the brain, we have searched through a good deal of English literature, and have collected the following cases:—

Case 1.—*Wilks and Moxon* mention, as an example of acute general cerebritis, the case of a young man, who was admitted into hospital supposed to be suffering from fever on account of the torpid state into which he had fallen; he never spoke, but lay perfectly motionless in bed, with his eyes open, apparently watching those around him. His illness lasted a few weeks; and after death the whole of his brain was found soft and pulpy, whilst some parts were semi-fluid.—"Pathological Anatomy," p. 237.

Case 2.—A man of 40, after frequent pain and throbbing in the L. side of the head, had convulsive paroxysms in the R. arm and leg, which were followed by giddiness, confusion of thought, and torpor of the R. side. Choreic movements were next succeeded by paralysis: speech gradually was lost, and also power over the sphincters; but the mental faculties seemed unaffected. He often screamed, as if in pain; and, after seven months' illness he became comatose, and died in three days.

"The L. hemisphere was soft and fluctuating throughout its whole extent, like a bag of fluid. On cutting into it there was about half-an-inch in thickness of sound cerebral substance; the remainder of the hemisphere was found nearly reduced to a fluid mass, partly consisting of purulent matter, and partly of cerebral substance in a soft pulpy state, but the greater part was purulent. From the mass of disease the ventricle was separated merely by the membrane which lines it, and contained a small quantity of serous fluid." There was a clot, the size of a walnut, in the left thalamus.—"Pathological and Practical Researches on Diseases of the Brain and Spinal Cord." 3rd edition. Abercrombie. Case 38, p. 94.

Case 3.—A complicated case. A middle-aged man one day had a fit for the first time in his life. The next day he fell in the street, and was picked up insensible, with a scalp wound. On rallying from a lengthened collapse he was very violent. For some days he suffered from intense pain in the head. On the seventh he became comatose and was trephined in consequence of an unfavourable appearance of the wound. He died the same night. *P.M.*—There was some serous effusion between the arachnoid and pia mater, especially at the base; the membranes were quite transparent; the ventricles were dilated and filled with a large quantity of fluid and the brain itself was watery throughout and soft. There was a small tubercular deposit as big as a pea, surrounded by a small area of softening and injection in one of the basal convolutions.—"Holmes' System of Surgery," vol. i., p. 623.

Case 4.—A boy, aged 13, died three weeks after he had been thrown from a donkey and fallen on the back of his head. The ventricles were enormously dilated and filled with serum and recently effused lymph, and the central white parts of the brain were very soft and broke down when slightly touched. The grey and white substance of the brain were throughout very much congested, and there was extensive inflammation of the membranes. There was a slight fissure of the occipital bone.—"Holmes' System," vol. i., p. 622.

Case 5.—A man, aged 37, died twenty-four hours after an injury of the head in a fall from a tree. The ventricles were found filled with fluid, and the fornix, remarkably soft, gave way when slightly touched. At the base of the brain around the pons and medulla oblongata ther was a large quantity of milky puriform fluid; the substance of the brain was firm, and the puncta of blood larger and more numerous than usual; the cribriform plate

of the ethmoid was broken and the corresponding part of the brain bruised.—"Holmes' System of Surgery," vol. i., 622.

Case 6.—*Wilks and Moxon* mention the case of a girl supposed to be hysterical. She was perfectly helpless, could scarcely move her arms, spoke and swallowed with difficulty, and had a stupid expression. After death the pia mater was found thickened, of a yellowish colour, and adherent to the brain, especially over the frontal lobes. There was found a general hardening of the brain, especially of the anterior lobes, which could be grasped like a hard tumour. The microscope showed the nerve fibres had almost disappeared, and their place was occupied with granule masses, vessels and fibrillated tissue.—"Pathological Anatomy," p. 238.

Case 7.—A girl of 14, in so helpless a condition of mind and body as to be at first mistaken for an idiot, had been an intelligent child a year before her death. She lay quiet in bed in a state of vacuity. At the *post-mortem* it was found she had been the subject of general meningo-cerebritis; the arachnoid was thick and opaque, and the pia adherent. The brain as a whole was much firmer than natural, and felt hard to the touch. No cause for this condition could be found.—Wilks and Moxon, "Pathol. Anat.," p. 238.

Case 8.—Dr. Bright in his medical reports relates the case of a little girl, who, for a year before her death, lay in a perfectly motionless and senseless state, with her limbs stiffly extended, and without the possibility of making the slightest movement. After death the white substance of the brain was found almost as hard as cartilage, and a stream of water washed off the grey matter, giving it the appearance of a wax model of the brain. The ventricles also looked as if they had been modelled in wax.—Wilks and Moxon, "Path. Anat." p. 238.

Case 9.—Dr. Henry Ashby records the case of a boy who died at the age of 20 months. Three days after birth he began with fits; when eight months old he was apparently an idiot, and blind and deaf, and the R. arm and leg were flexed and more or less stiff. The rigidity increased; the symptoms of descending degeneration were more marked on the right side than on the left. He lay in bed with head retracted and face turned to the L. side. At the necropsy the arachnoid was thickened and opaque, and the pia consisted of many tortuous vessels. There was no optic neuritis or atrophy. The brain substance was remarkably hard and firm, and apparently shrunken. That between the cortical grey matter and the basal ganglia was firm

and dark in colour, and evidently sclerosed. The lateral ventricles were much dilated. There were descending changes in the cord and medulla. The microscope showed the neuroglia to be exceedingly coarse and granular, the perivascular spaces dilated, and in places an increased amount of fibre tissue accompanying the vessels in their course.

Dr. Ashby says the most plausible explanation of this condition of sclerosis is that it is due to meningo-encephalitis of the convexity, which took place during fœtal life, and which was followed by atrophy of the convolutions and descending sclerosis.—*Pathological Society's Transactions*, vol. xxxvii., p. 12.[1]

These nine cases present us with two distinct forms of encephalitis. The chief pathological feature in the one is *softening*, and in the *other hardening* of the brain tissue, but in all the changes are *diffuse*.

In the cases in which there was softening (1 to 5), and in which the inflammation was more or less acute, the changes involved either the whole brain, or one hemisphere, or central softening was accompanied by increased vascularity of the remainder.

It is, further, to be noted that in one of the cases where the latter condition was found, 24 hours only had elapsed between the injury and death, and the membranes at the base were presumably affected. In the other, after three weeks, in addition to a very marked congestion of the unsoftened parts, there was extensive inflammation of the membranes.

The *duration* of the disease varied from 24 hours to a a few weeks, but in one case, with peculiar features, it was seven months.

Traumatism was the exciting cause of the inflammation in three cases (3, 4, 5,) but in only one (5), was it probable that the membranes had been penetrated, and in that the softening was found in the central parts; and in another (3) it is uncertain what predisposing effect a small previously existing tubercular focus may have exerted.

[1] Two cases are recorded in "Fagge's Medicine," p. 554, vol. i. One is probably a fuller account of Case 7. The other is the case of an infant who had an illness of 14 months, marked by convulsions, followed by long periods of unconsciousness. The hemispheres were indurated and the pia adherent.

In the other two cases there is nothing to suggest the cause of the disease.

In case 2, though the pathological appearances were those of a diffuse encephalitis the clinical history suggests that the disease took a course very different to what occurred in the others. The way in which additional symptoms slowly made their appearance, impairment of function preceding its abolition, point to a gradual implication of portion after portion of brain until the whole hemisphere was converted into a semi-purulent mass. Such a condition from the chronicity of its course, and the formation of pus presents some likeness to a cold abscess, and it serves to illustrate the difficulty of deciding in some instances to which category, acute or chronic, localised or diffuse, a particular case is to be relegated.

In cases 6, 7, 8, 9, *there was a general hardening* of the whole brain, and in three of them, at least, the membranes had been the subject of a chronic inflammation.

The disease in all was probably identical, and the hardening due to an increase in the fibrillated tissue throughout the organ and along the course of the vessels.

In the three cases in which the *duration* is stated or can be inferred, the disease lasted from 12 to 20 months.

In no case is there any history of traumatism, and in all the cause was obscure.

The patients were all young, probably all under 20.

In Ashby's case the disease originated as an intra-uterine condition, and this is the only instance in which the records, we have had access to, note the occurrence of convulsions.

In the only case in which the state of the optic discs is mentioned it was normal, though the infant was thought to be blind.

The most prominent symptom, and one which was present in all these cases, was the *state of vacuity and helplessness* in which the patients lay for long periods. The impairment and abolition of function was evidenced in the loss of intelligence and muscular power more or less complete. We believe that *this long continued torpor of mind and body* is the most salient feature of chronic diffuse encephalitis.

Allusion has already been made to the fact that in the chronic cases with hardening, the whole brain was affected.

In the acute form, if the patient survives long enough, the same torpor may be present when the whole brain is involved—(case 1); but in acute cases of short duration, this valuable symptom may never develop, as signs of concussion or compression are likely to predominate from the first.

Therefore, though this condition (torpor of mind and body) may be present in acute diffuse encephalitis, it is more especially characteristic of the chronic form in which induration is the chief pathological feature, and it is, then, usually of long duration.

The symptoms due to secondary degeneration in the cord call for no remark.

THE PHYSICS OF THE CRANIAL CIRCULATION AND THE PATHOLOGY OF HEADACHE, EPILEPSY, AND MENTAL DEPRESSION.

BY A. HAIG, M.A., M.D.OXON., F.R.C.P.

Physician to the Metropolitan Hospital and to the Royal Hospital for Children and Women.

In the Spring number of BRAIN, 1891, I pointed out that certain cases of headache, epilepsy and mental depression bore what appeared to me to be an important and fairly constant relation to the excretion of uric acid in the urine.

I suggested that an excess of uric acid relatively to the urea in the urine was an index of an excess of urate in the blood, and that such excess of urate, by contracting the arterioles and capillaries might produce cold skin and extremities and scanty urine, and by effecting similar changes in the intracranial circulation might produce the headache, the fits, or the mental depression.

Further than this, I did not at that time attempt to go: but I believe that it is now possible not only to bring forward further proof that urates have the action on the vessels which I then attributed to them; but that this vascular effect of urates being granted it is now no very difficult matter to show by the aid of the researches of others that the changes in the intracranial circulation which urates may thus bring about may be quite sufficient to produce headache, fits, or mental depression.

The subject, therefore, naturally divides itself into two parts. (1) The proofs that urates do contract the arterioles and produce high arterial tension. (2) The effects of such high arterial tension on the intracranial circulation, and to these I shall add: (3) A short review of the symptoms

and pathology of these disturbances regarded from this point of view.

As regards the effects of urates on the circulation I have previously mentioned that in migraine the pulse is well known to be slow and of high tension, while the urine is scanty and the skin and extremities cold, and some six or seven years ago I pointed out that the urine excreted during an attack of severe headache contains a more or less large excess of uric acid relative to the urea excited at the same time, and I suggested that the excess in the urine was the index of a relative excess of urate in the blood.

I further pointed out that by attending to certain laws that appeared to govern the solubility of urates it was possible to increase or diminish at pleasure the excretion of urates in the urine, and that along with such increased excretion, however produced, went high tension and slow pulse, cold skin and extremities, scanty urine, mental lethargy and depression, or headache; and along with diminished excretion, however brought about, there went quick and relatively low tension pulse, profuse urine, warm skin and extremities, mental brightness and well being; so that it is possible, as I have said, to influence the pulse rate and tension, and the mental condition to almost any required extent, and did time and space permit I could now bring forward scores of cases in which I have done this, and not a few in which others have been able to do the same.

See among others Dr. J. T. Eskridge in the *Alienist and Neurologist*, Jan., 1892, for some interesting cases of headache, depression, and hysteria, where drugs and other treatment acted just in the way that I have described.

But there is a further fact which now testifies to the truth of my reasoning, for not only is the urine scanty in the uric acid headache, and profuse when it passes off, and the same in epilepsy and hysteria, that is to say, the excretion of water varies in these diseases inversely as the uric acid, when the urate excretion is high the water is low, and *vice versa*; but the very same thing holds in physiology also; so that in every one and at all times the urinary water is inversely as the urate excreted along with it.

If this is a fact (a physiological fact) it is alone sufficient to prove the influence of urates on the vessels, for what other possible explanation can be given of the constant relation of the excretion of urates to the excretion of water.

Further, in a recent paper in the *Lancet* (July 9th, 1892), "On the elimination of water and the pathology of dropsy," was able to show that this law of the inverse relation of urates to water dominated and explained a whole host of well-known facts in therapeutics and pathology.

That in therapeutics all drugs which clear urates out of the blood and diminish their excretion in the urine do at the same time relax the arterioles, and produce a diuresis, and that the well known diuretic action of such drugs as mercury, opium, iodide of potassium, and other iodides, the salts of lithium, and the salicylates, etc., is a simple illustration of this law, which completely explains their action.

And conversely that drugs which increase the urate in the blood and urine, such as the alkaline salts of sodium, and potassium, the salicylates and the neutral phosphates, do, while the increased excretion of urates lasts, produce contracted arterioles, slow high tension pulse and scanty urine.

I also pointed out that in pathology the same law holds, that in all diseased conditions which tend to bring an excess of urate into the circulation or retain it there, we have contracted arterioles, slow high tension pulse, and scanty urine. Among such conditions I have specially mentioned migraine, some cases of epilepsy, certain menstrual troubles, especially dysmenorrhœa, many conditions of low health and nutrition, as the failure of nutrition in old age, and after disease; Bright's disease, both acute and chronic, Raynand's disease and paroxysmal hamoglobin-uria (see *Transactions of the Medical Society of London*, vol. xv., and *St. Bartholomew's Hospital Reports*, vol. xxviii.). In all these conditions we have slow high tension pulse and scanty urine, and according to the way in which the high tension affects the intracranial circulation in a given case, we have head-ache, mental depression or fits (epileptic or hysterical), and other manifestations of epilepsy or hysteria, not to mention syncope, cerebral hæmorrhage, and a host of other structural and nutritional alterations.

Conversely the pathological condition (fever) which most quickly clears the blood of urates and diminishes their excretion in the urine (driving it, as we often know to our cost, into the fibrous tissues, the joints, liver, spleen, &c.) does at the same time relax the arterioles, producing quick low tension pulse, and at its first outset a more or less marked diuresis.

So much is this the case that I offered (*Lancet*, prev. ref.) to tell the days in any chronic hospital case on which the temperature had risen above normal simply from the record of the urine passed, if no drugs affecting the problem were being given.

I assert, therefore, without fear of contradiction, that in physiology, pathology, and experimental work in man, the urinary water is from hour to hour, or from day to day, inversely as the uric acid excreted along with it; and that drugs which increase the excretion of urate diminish the water and *vice versa*.

The pulse rate and tension bear also precisely the same relation to the urate excretion, and they do this because the urate excretion is to a large extent an index of the amount of urate in the blood (Bright's disease being an exception to this rule for reasons given elsewhere; see "Uric Acid," J. and A. Churchill, 1892, p. 225.)

The one series of facts explains the other; the water is scanty because the arterioles are contracted, and I have pointed out that Dr. Lauder Brunton has shown that drugs like digitalis, which contract the arterioles, may diminish or even completely stop the flow of urine (see "Uric Acid," p. 100.)

The heart's action is slowed because the contracted arterioles raise the tension, for Marey has formulated the law that the pulse rate is inversely as the arterial tension.

There is an exception to this law which is also frequently met with in Bright's disease, for contracted arterioles and high tension in this disease are not always accompanied by a slow pulse, and the explanation of this is, I think, as follows:—Marey's law, I believe, holds good so long as the heart is up to its work, so long as its systoles are complete,

but if the heart begins to give way, and the ventricle dilates, its systoles are imperfect and must be repeated more often to keep up the circulation.

Thus, in acute Bright's disease with heart failure, or at the end of a chronic case as dropsy comes on, you find in the heart increased area of dulness, apex to the left, 1st sound reduplicated, and 2nd sound loud, but the pulse instead of being slow is 85 to 90 in the minute; here we have cardiac failure with dilatation.

And there is this marked difference between the slow and the quick pulse in Bright's disease, as regards the effect of drugs.

Take a case of Bright's disease in which the heart is strong and compensation is perfect, there are all the cardiac and vascular signs of high tension, and the pulse is slow, say 45 to 55. Give any of the drugs which clear urates out of the blood, acids, the nitrites (which act as acids, see "Uric Acid," pp. 40-41), opium, mercury, or iodides, you relax the arterioles, increase the urine, and generally (in my experience) diminish the absolute quantity of albumen, and the pulse will quicken up to 70 or 80.

But take a case of Bright's disease, in which owing to cardiac failure, the pulse is quick; give any of the above drugs as before to relax the arterioles; you will increase the urine and diminish the albumen as before, but as you do this the pulse will get slower, instead of quicker. As you relieve the heart from the load of high tension against which it had to work, its systoles become more complete, and it alters its rate of action in the direction of the normal, that is, the pulse becomes slower instead of, as in the other case, faster.

With regard to my statement as to the inverse relation of urates and urinary water there are a few things which might appear to anyone taking up the subject *de novo*, to be exceptions, when they are not so in reality.

Thus it is obvious that relaxing the arterioles cannot cause diuresis when there is no available water in the body, and conversely when, as in dropsy there is a great excess of water in the body, relaxing the arterioles will cause great and lasting diuresis, which continues till the dropsy is all gone (see interesting case mentioned in "Uric Acid," p. 225).

But to return to the other condition, there may be no available water because profuse perspiration, diarrhœa, or previous diuresis has removed it all.

Thus a diuresis, at any given time, is generally due to two factors: 1st, that the urates have been cleared out of the blood, and the arterioles relaxed as a consequence of this, and 2nd, that some previous contraction of arterioles has kept back and retained in the body some water which now has an opportunity of passing out.

I have often previously mentioned the fact that in physiological conditions the water is least in the day, say 40-50cc. per hour, and most in the night, as 70 to 80cc. per hour, this being just the reverse of the urate excretion; but the diuresis in the night is the result of the retention of water during the day, and is generally proportioned to it: *i.e.*, if there is a day of more than usual high tension with it results headache, mental depression and scanty urine, and if at night the arterioles are relaxed and the tension falls the urine will be more than ordinarily profuse, being double the hourly excretion of the day, and far more per hour than on any night corresponding to a day of ordinary tension.

In Bright's disease this is nearly always the case, and the urine of the 12 hours ending 8.0 a.m. is very often double that of the 12 hours ending 8.0 p.m. In other words, the urine in chronic Bright's disease is only profuse at night; by day the tension is high and the urine scanty.

Further, by altering the time of the urate excretion, one can alter that of the water excretion also; thus let anyone take such a dose of alkali (not lithia, for reasons given in "Uric Acid," p. 29) as shall render his urine alkaline all through the 24 hours, and continue this for seven to eight days. After two or three days, he will find that the urine is now much the same in quantity per hour both night and day, that it is not scanty during the day and morning hours, and therefore not profuse at night.

And the reason of this is obvious: the plus excretion of urate which normally takes place in the "alkaline tide" of the day is the result of the retention of urate during the acid tide of the previous night, but he has been taking alkali for

several days, and there is now no acid tide of the night, and its effects have disappeared with it; there is now little or no retention of urate at night, hence there is no plus excretion of urate in the alkaline tide next day. As there is no plus excretion of urate by day, its effects on the arterioles and the excretion of water are absent; there is no excess of it in the blood, no holding back of water, and, consequently, no diuresis the following night.

Indeed, under these circumstances, the urine may be quite profuse by day, and then it is scanty at night, because all the available water has been run off by day; this being just the reverse of what takes place when the acidity is normal.

This is only one instance of the way in which, by controlling the urate, we can control the water, and, at the same time, alter also at pleasure the rate of the heart's action and the tension of the pulse.

From these and many other facts which there is no space to give in full here, I infer and assert without fear that, other things being equal, the contraction of the peripheral arterioles throughout the body is directly proportional to the amount of urate that is circulating in the blood.

I do not assert that there are no other substances which produce the same effect, but I believe that in the human body urates are more important than any other substances or combination of substances, and this is the reason of the position which I have found them holding in relation to certain disease processes about which I have written.

Let it be granted, then, that contraction of the arterioles is proportional to the urate in circulation; we can now pass on to our second problem, viz., how does the high tension produced by this contraction of arterioles affect the intra-cranial circulation, and how may these effects help us to elucidate the pathology of those cerebral disturbances, which I have found by observation to bear a special relation in time to the excretion of uric acid?

While thinking over this problem, I became greatly interested in some articles on the intra-cranial circulation, which appeared in "Virchow's Archives," by Dr. Geigel (*Archives*, vol. cxix.), Prof. Lewy (*Archives*, vol. cxxii.), and

an account of an experimental investigation by Prof. Dr. H. Grashey ("Fest Schrift München," 1892).

I shall mention these here, however, only so far as they appear to me to bear on our problem.

Geigel's main contention is that in the conditions which obtain within the skull dilatation of an artery, does not increase the flow of blood through the brain, but really diminishes it, because it compresses the veins and capillaries.

Lewy dissents from this in so far as it refers to physiological conditions, as he points out that in these conditions the lymph channels and capillaries are so numerous that they will bear a good deal of compression before they really diminish the blood flow through them, but he acknowledges that in disease, when part of the available space has been already taken up, say, by a cerebral tumour, or where some of the available capillaries have been blocked or destroyed by meningitis, it might then be, as Geigel asserts, that dilatation of an artery might actually diminish the quantity of blood flowing through the brain.

Both these writers agree with each other and with a previous writer (Althann), whom they quote, that it is not important how much blood is present in the skull; but how much flows through the brain in a given time, because on this depends that satisfactory interchange between the blood and the tissues on which the functions of the brain are so absolutely dependent.

Grashey appears to believe in this also, but his investigations are in great part directed to the determination of the effects on the intra-cranial circulation of increased arterial pressure, and bear therefore in the most direct manner upon our problem.

He tells us (previous reference, p. 73) that as regards the nourishment of the brain, it is, apart from the quality of the arterial blood, of more importance how much streams through the cerebral vessels in the given time.

He then points out that his researches show that, other things being equal, the quantity of blood which streams through the cerebrum in a given time rises with the pressure in the aorta up to a certain point, but when that point is

overpassed the intra-cranial pressure is so great that the central veins are compressed and begin to vibrate, and with this vibration there is always a considerable reduction in the amount of blood passing through ; and though the veins and capillaries behind the point of vibration are overfilled with blood, the conditions become unfavourable to the nutrition of the brain.

But, he continues, since the cerebro-spinal fluid comes from the blood, it is not improbable that the stasis in the veins and capillaries thus brought about may result in an increase of its quantity, and so we get a further increase of intra-cranial pressure, so that when once the pressure rises so that the central veins begin to vibrate, a vicious circle comes into existence, which must bring danger to the brain.

We shall see, I think, further on that this suggestion of Grashey's with regard to the effects of high pressure and stasis on the quantity of the cerebro-spinal fluid may help us to explain some points in the pathology of uræmia.

He thinks that the veins are specially compressed at their point of entry into the cerebral sinuses, and he does not believe that high pressure compresses the capillaries ; on the contrary, they are overfilled with blood, owing to back pressure from the veins.

We have, then, as the result of these researches and my own, certain facts as observed, and what seems to me a very simple explanation of their inter-relation and causation.

The facts are those observed by myself, and mentioned in my previous paper and elsewhere, namely, that certain forms of headache, epilepsy, and mentral depression bear an important relation in time to the excretion of urates in the urine, and that by controlling the formation and excretion of urates, these disturbances can also, to a large extent, be controlled.

I have explained these facts by suggesting that the excessive excretion of urates in the urine is the sign of a similar excess in the blood, and that when we alter the amount in the urine we at the same time alter the amount in the blood ; and have suggested that the relation of these disturbances to each other and to the excretion of urates was due to the fact that excess of urates in the blood affected

the intra-cranial circulation, and so altered the functions of the brain.

It appears now from the interesting researches of Professor Grashey that we can go a step further and point out the exact way in which urates act on the intra-cranial circulation, namely, by producing high arterial tension.

The effects of uric-acidæmia on the skin and extremities, causing them to be cold and pale, is easily explained by the contraction of arterioles and capillaries which it produces in these parts. The same effect on the arterioles of the kidney may also suffice to explain the scanty urine of uric acidæmia. But we are met by a difficulty when we come to apply the same reasoning to the intra-cranial circulation, for it has been pointed out by Professor Horsley ("Origin and Seat of Epileptic Disturbance," *British Medical Journal*, vol. i., 1892, p. 63), that his experiments all go to show that the condition of the cortex during the epileptic attack is one of congestion, not of anæmia.

It appears then that if the uric acidæmia is the cause of headache, epilepsy, and mental depression, that it must act on the brain in a somewhat different manner from that in which it acts on the skin and kidneys.

And Professor Grashey's researches suggest at once an explanation of the difficulty.

Uric acid, when it contracts the arterioles of the skin, kidneys, and other organs, not only affects these organs, but produces a general effect throughout the whole body, namely, high arterial tension; this high arterial tension will, doubtless, affect the intra-cranial circulation in the manner that Grashey describes, and, producing more or less stasis there, will quite account for the headache, epilepsy, and mental depression, and their time relation to an excessive excretion of urates.

This also explains completely Professor Horsley's observation, for the intra-cranial condition is one of stasis and hyperæmia, not of anæmia, and the stasis and hyperæmia are due to the compression of the veins by the high pressure in the arterial system, so that we have high pressure between the tense arteries behind and the compressed veins in front,

XUM

and with this stasis and deficient interchange between the blood and the tissues.

Our chain of causation will then run as follows :—(1) Uric acidæmia due to very numerous causes mentioned elsewhere, which are, however, to a large extent, under our control; (2) general contraction of arterioles and capillaries producing high arterial tension; (3) the results of the high tension, more or less stasis and hyperæmia in the intra-cranial circulation, producing in accordance with the various anatomical and physiological conditions present in a given case, headache, mental depression, or fits.

It will be found, I think, that this completely explains the observed relation in time between the excretion of urates and these functional disturbances, and will also enable us to explain more completely than has previously been possible a whole series of points in their causation and relation to other diseases.

The relation of migraine, or as I have called it the uric acid headache, to uric acid is as follows :

It is contemporaneous with a large excretion of uric acid in the urine having a relation to Urea of 1-18 or 1-20, the absolute hourly excretion is also large, say .6-.7 gr. per hour = to 14-17 grs. in the 24 hours, in a person whose normal excretion in the 24 hours would be from 10-12 grs. When a headache has gone, or has been driven off by drugs, it will be found that the hourly excretion has fallen to about .4grs. per hour.

Again, the plus excretion which accompanies the headache is preceded by a minus excretion, and if a line be drawn to represent the normal relation of uric acid to Urea in excretion at 1 to 33, it will be found that the excursion below that line which precedes the headache is almost exactly equal to the excursion above the line which accompanies the headache.

In a word there is a fluctuation in the excretion of urates, a minus excretion preceding the headache, and a corresponding plus excretion accompanying it.

Now it is quite impossible to prevent these fluctuations in the excretion of urates; they occur in everyone, and

almost every hour of the day, as the result of a whole host of causes which it is impossible to shut out; is it then impossible to prevent these headaches?

This does not necessarily follow, for the headache does not depend on the fact that there is a fluctuation in the excretion of urates, but only on the size of that fluctuation.

Supposing, as I have done for reasons given elsewhere (see "Uric Acid," p. 12) that all the uric acid which is in excess of the relation to Urea of 1 to 33 passes through the blood, it is obvious that an excretion of 1 to 20, on an average urea formation (and it may be considered that the urea excretion generally equals its formation, because it is soluble) of 600 grs. will furnish 12 grs. of uric acid to pass through the blood; while an excretion of 1-20 on a urea formation of 300 grs. would only furnish 6 grs. of uric acid to pass through the blood.

By reducing our whole nitrogeneous metabolism it is thus obviously possible to prevent any large amount of urate from ever, in physiological conditions, finding its way into the blood, and if the headache is due to the absolute quantity of urate in the blood it must thereby be prevented.

Now this is exactly what I have done. I have reduced my average urea formation from 500 grs. or more which it used to be when I took ordinary diet, to 350 grs. a day now that milk and fish in small quantities form the only animal food.

Contemporaneously with this change my headaches fell in frequency and severity, and in 1891, I had only 5, and in nine months of this year only 3; while on ordinary diet they used to range as high as 40-50 in a year.

And that this change is due to the change of diet is evidenced, not only by the fact that the two changes were contemporaneous, but by the further fact that if I alter my diet and increase my urea I promptly begin to suffer again, and, indeed, I believe that but for occasional and unavoidable indulgence in meat, I should now never have a headache at all.

And there is one further fact, namely, that every single drug (and there are at least a dozen of them that are well-known) that diminishes the excretion of urates in the urine, at the

XUM

same time relieves or cures the headache, and the headache is proportional, not to the amount of the drug given; but to the effect of that drug on the urate excretion.

It is, therefore, never safe to say this drug is no use. I have given so many grains of it, and the headache continues; examine the urine and you will see that the large excretion of urate continues also; either your drug has not been absorbed owing to a sick stomach, or its effect on the solubility of urate has been counteracted by some other condition present, as for instance a large amount of alkaline phosphate or other alkaline salt in the blood.

Such is the relation of this headache to uric acid, and it has long appeared to me that the only possible explanation of these facts is the one I have given, that the excess of uric acid in the blood (uric-acidæmia) produces the headache.

I think we can now see pretty clearly the way in which it effects this result.

I will also say shortly that a very considerable number of cases of epilepsy, hysteria, mental depression and irritability bear precisely the same relation to the excretion of urates that the headache does, and they also are similarly relieved or cured by drugs or diet that tend to clear the blood and urine of urates, or diminish its formation.

The great point in the diagnosis of uric acid disease is that it is temporary and fluctuating in accordance with the fluctuations in urate excretion, and is not permanent and continuous.

If a headache lasts day after day for a week and does not vary with the natural variations in the excretion of urates, or such artificial variations as can be produced by drugs, it is not due to uric acid, unless Bright's or other organic disease is also present, when we may have an organic and not a functional uric acidæmia.

If fits, mental depression, or irritability, are similarly without fluctuation in accordance with urate excretion, they also are probably not connected with it, and search should be made for organic disease.

I have shown above that contracted arterioles and capillaries, and the high arterial tension they produce, bear

exactly the same relation to the excretion of urates and the fluctuation that can be produced in this by drugs and disease as the above functional disorders do; and the reason of this is that the high arterial tension is due to the uric acidæmia, and this in its turn is the cause of such serious disturbances of the intra-cranial circulation as may easily account for the origin of these functional disturbances of the nervous system, and even more serious troubles.

If, then, it be true, that certain cases of headache, mental depression, and fits, are due to the effects of high arterial tension on the intra-cranial circulation, those things which tend to raise arterial tension will do harm, and those that tend to reduce it will do good. Let us apply this rule to our knowledge and experience of the things that seem to condition these disturbances.

Now high arterial tension varies, chiefly with two factors (1) the contraction of the arterioles and capillaries, and (2) the power of the left ventricle of the heart to overcome the intra-arterial pressure at each systole.

Therefore, arterial tension can be lowered by two things, namely, anything that will relax the arterioles and capillaries, or anything that will weaken the left ventricle and prevent it discharging the whole of its contents into the aorta at each systole.

A great rise in arterial tension is produced as we know also by muscular exertion, though the effect is only temporary, and a great fall in arterial tension can be produced in a given region by compressing the main artery of that region.

Now it is a characteristic feature of the uric acid headache that it is worse on assuming a stooping posture and on making any muscular exertion or effort; but if it is due to anæmia of the brain it is not very easy to see how either of those acts can increase the anæmia. On the other hand, if it is due to high arterial tension compressing the veins, it is clear that muscular exertion which raises the tension will do harm, while the stooping posture will facilitate the inrush of arterial blood, but still further impede the outflow of the venous blood.

Further, it is obvious that pressure on one or both arteries

in the neck will reduce the arterial tension in the skull, and so relieve the pressure and pain, which in my experience, it at once does, while if the disturbance were due to anæmia it must obviously increase it.

But muscular exertion has another effect if it is persevered in, for it produces relaxation of arterioles and a fall of arterial tension, and I have been told by numerous sufferers (and have myself also often tested the same point) that though exertion at first makes the headache much worse it eventually cures it if persevered with. Let any sufferer put on his hat and go for a sharp walk; his headache will be much worse at first, but at the end of say half an hour, he will have got thoroughly warm, his arterioles will be everywhere relaxed and his pain will have vanished, and he will once more be able to take a cheerful view of things.

This same relaxation of arterioles relieves at once both headache, mental depression and irritablity, for all are due to the same high tension, and which of them will be most marked in any given case depends to some extent on the height of the tension, but also probably on the anatomical conditions, which influence in one way or the other the effect of the high tension on the intra-cranial circulation.

Dr. Liveing tells us of the interesting results obtained by Dr. Parry in cases of this headache, also in hysteria, epilepsy and mania, by compressing the carotid, and in some cases he instructed the patients how to perform this operation for themselves. ("Megrim and Sick-headache," pp. 276-7.)

On this theory also we can see at a glance how venous congestion produced by coughing, etc., may still further impede the outflow of blood from the intra-cranial veins and produce vertigo fits and other members of this group.

When my headache is bad it is simply impossible to assume the horizontal position, and when in bed I have to sit bolt upright against the back and slide gradually down if the pain gets less. I have also pointed out that by turning the face up so as to look at the ceiling the pain is somewhat relieved. It seems to me that this position may counteract to some extent the arterial inrush, especially from the vertebral arteries, and may also facilitate the outflow of venous blood

by the jugulars, but I should like to hear the opinion of a more competent anatomist on these points.

There is probably no fact that is more widely recognised in this portion of the globe, than that mental irritability and depression are relieved by the smoking of tobacco, and if we look up tobacco in a system of therapeutics we find that it is classed with the depressomotors, *i.e.*, with the drugs that diminish muscular power and enfeeble the heart, and among drugs having a similar action it is interesting to note that ipecacuan and antimony have a similar power in relieving migraine. Now, how do they act? It seems to me that tobacco relieves mental irritability and depression, not so much by relaxing the arterioles, though its effect on the muscles may also do something in this direction, as by weakening muscle power generally, and especially that of the left ventricle of the heart, it thus reduces arterial tension and relieves the intra-cranial circulation, and we know that if tobacco is pushed beyond the stage of mental well-being it may cause nausea, and very serious depression or syncope.

My observations on the effects of tobacco smoking on the pulse seem to show that it lowers arterial tension, and quickens the rate of the heart's action, and it is easy to understand that in accordance with Grashey's observations it will in this way increase the volume of blood passing through the brain, and so produce well-being; and this well-being produced by tobacco is not accompanied by a desire for activity and a feeling of tone in the muscles, but rather by slight languor and disinclination for exertion.

I think also that it is of great interest to note that a physiological condition which has a similar effect on the heart has also a similar effect on the uric acid headache; I refer to old age, for it is well known that as sufferers begin to grow old this headache gets less frequent, or even entirely ceases.

The explanation is, I think, fairly simple, and I have been fortunate enough to see or hear of several cases of old people who have lost their headaches in old age, after being regular periodical sufferers all their lives. But a closer investigation of such a case brought out the following interest-

XUM

ing facts: the headache indeed was absent, but the rest of the phenomena were not absent, just as for so many years before there came a time when the urine was scanty, followed shortly by a time when the urine was profuse, with the scanty urine went coldness of skin and extremities and some mental depression, some rise of arterial tension, but in place of a long slow pulse a fast and irregular one, and a palpitating heart.

This sufficed to explain everything; of uric acidæmia and an increased excretion of urates we had ample evidence—it was not that they were absent, but that their effect had altered.

In place of a long slow pulse we had a short and fast one and a palpitating heart, the heart was failing—no uncommon occurrence in old age—and owing to this failure the intra-arterial pressure, in spite of contracted and degenerate arterioles, was not great enough to compress the intra-cranial veins and produce headache; all the other factors were present as before, the heart and head signs alone had altered, and the alteration of the former explained the alteration of the latter.

In a similar way, and doubtless for similar reasons, mental depression and irritable temper often get decidedly less marked as old age advances.

As a rule in the young and middle aged, whose hearts are intact and well nourished, the severity of the symptoms is proportional to the amount of urate that is passing through the blood and being excreted in the urine.

Thus with an excretion of .6 gr. per hour, there will be mental irritability, mental and bodily inability and depression; with an excretion of .7 gr. per hour and above there will in the same person be headache or fits—whichever he happens to be liable to, and in practice we often see mental depression and irritability forming a sort of halo round the other more severe disturbances of function.

In my own case, though the amount of urate passing through my blood is now rarely sufficient to produce a headache, I much more often suffer from some amount of irritability and depression.

Another instance of the same thing may be seen in post

febrile bradycardia and the depression which follows acute disease, due, I believe, to post febrile uric-acidæmia.

Writers on post febrile bradycardia (see "Uric Acid," pp. 117 and 135) have observed quite correctly that this condition is met with in its most severe forms in those who have been previously strong and healthy, and yet in apparent contradiction they attribute the bradycardia to weakness of the heart muscle.

Why should those who were previously strong and healthy have the weakest hearts after a fever? There is no difficulty in showing, however, that they have most uric acidæmia, and the post febrile phenomena are proportional to this.

In my book ("Uric Acid," p. 135), I have mentioned two cases of pneumonia that were under my care about the same time: one was a strong and powerful man, the other a weak and ill-nourished woman; in the former the post febrile conditions, including bradycardia, were very prominent, in the latter very slight, and the difference was simply due to the fact that the big man with his large metabolism of nitrogen stored much urate at the time of the fever, while the ill-nourished woman stored but little, and the post febrile symptoms were proportional in each case to the amount of urate passing through the blood (post febrile uric-acid-æmia).

As regards the heart, the only necessary quality for the production of bradycardia is that it shall be sufficiently strong to withstand the strain which uric acidæmia, contracted arterioles, and high arterial tension put upon it, and this power is, to my mind, much more likely to be met with in those who were previously strong and robust.

If, on the other hand, the heart fails before the high tension, we get palpitation and a quick and irregular pulse marking the period of post febrile uric acidæmia, just as in the case of headache previously mentioned.

During our recent epidemic of influenza, I have several times been struck with the fact that the old and feeble, when they recover from uncomplicated influenza, have far less after depression than those in the prime of life, and I have no

XUM

doubt that their small nitrogenous metabolism, as in the case of the woman just mentioned, accounts for this, and I have little or no doubt that the severe depression which follows influenza is simply a prolonged post febrile uric acid-æmia, which, if not actively treated, may lead the way to more or less permanent feeble nutrition.

The old, again, are probably let off some of the post febrile mental depression because their hearts are unable to maintain the arterial tension necessary to interfere with their intra-cranial circulation, and their comparative immunity is thus due to two causes.

But the absolute dependence of all the phenomena on the amount of urate present can easily be demonstrated in physiological conditions.

Let anyone who does not mind a little auto-experimentation take, say, gr. xl. of an alkaline salt of sodium or potassium three times a day, and continue this for several days; during the first two or three days as the acidity falls the excretion of urates will rise, the urine will become scanty, and he will suffer from headache, mental depression, and coldness. Let him continue the same dose for another two or three days, he will find that the urine gradually becomes more profuse, and that the headache and mental depression disappear, and feelings of well-being take their place. But the alkali is still being taken. What is the alteration due to? He will find that the urate excretion has altered; it was at first very high, but after two or three days the immediately available stores were exhausted, and the excretion fell down nearly to formation level (*i.e.*, 1.33), and little or no urate passed through the blood. This is the cause of all the other alterations, the alkali itself having no direct effect whatever.

Or the experiment can be varied by taking salicylate of soda, gr. xv. quater for two or three days, and following it by the alkali as before.

The salicylate is a powerful solvent, and quickly clears out all the available stores of urate. The alkali given in sequence to it will cause but a comparatively small excretion of urates, and the symptoms it produced on the previous occasion will be more or less completely absent.

Having taken the view that, in accordance with the above facts, mental depression bears exactly the same relation to the excretion of urates that headache does, and that the causation of the one differs from the causation of the other merely in degree, I was much interested to be told by one of my patients (a man of conspicuous mental ability, who had all his life suffered from mental depression), that when he had his worst attacks, which were always in the early hours of the morning (note the parallel with the normal excretion of urates, which is always large in the early morning hours—early alkaline tide—and the urine scanty, though, as I have shown, there is plenty of water in the body, and no digestion is going on[1]), he was accustomed to obtain relief by sitting upright in bed in exactly the position that I have to assume in my headache ; and the fact is not only interesting as a parallel, but it proves, I think, that in this case the mental depression was not due either to feeble heart or to cerebral anæmia, and taken along with my argument about the effects of tobacco and cardiac depressants, explains the frequent association of high arterial tension with mental depression, because, as we now know, they are cause and effect.

I do not doubt that mental depression may be due to a weak heart, and consequent cerebral anæmia, but such depression would pay little or no attention to fluctuations in urate excretion, and would be benefited rather by lying down than by sitting up.

It is interesting also to remember that in mental depression associated with high arterial tension, digitalis does harm. I have several times given it in cases where there seemed to be a doubt as to the power of the heart, and have seen cases where others have done the same, and the only result was to make the depression worse.

One can easily understand why this should be so, as digitalis affects both factors in the causation of high tension : it strengthens the heart and contracts the arterioles. On the other hand, where mental depression is associated with

[1] See also Proceedings of the Medical Society of the State of Pennsylvania, June, 1890, as to early morning hours in melancholia.

XUM

real cardiac weakness and low tension, digitalis may do a great deal of good.

Talking of mental ability reminds me of a point of some interest in this connection.

Dr. Liveing speaks of the person whom he would expect to be affected with megrim as "a man of high intellectual attainments" (prev. ref., p. 35), and Dr. Wilks [1] considers that megrim is rather rare among the lower orders, and though I have seen a good number of cases among hospital out-patients, I do not recollect to have seen any among the lowest class, the ordinary day labourer.

There seems, then, to be some reason to think that megrim is more often met with among those who have naturally active brains, and if this activity has as its anatomical basis the possibility of a free supply of arterial blood, it is not difficult to see that those who have the largest arteries will be most likely to have their intra-cranial veins compressed when the intra-arterial tension rises too high.

I have mentioned above Grashey's supposition that one of the effects of increased intra-cranial pressure and stasis might be an increase of cerebro-spinal fluid which might further increase the pressure, or keep it up even after the arterial tension had fallen.

Now Dr. Broadbent,[2] whose opinion I have several times quoted before, considers that the phenomena of uræmia represent the effects of high tension on the circulation of the brain, and Grashey's researches make it easy for us to understand the way in which it acts.

But in accepting this explanation I have had considerable difficulty in understanding how it was that when, in some such cases, the temperature rose, this being almost certainly accompanied by a relaxation of arterioles and a fall in arterial tension, the cerebral symptoms, coma, &c., remained unaltered, *i.e.*, that the cause being removed, the effect still continued; but if one of the effects of the high tension is to bring about an increase of cerebro-spinal fluid, it is quite

[1] *Lancet*, vol. ii., 1888, p. 263.
[2] "The Pulse," p. 245.

possible that so much pressure may be produced in this way, that the fall of arterial tension, due to the rise of temperature, is no longer sufficient to relieve the intra-cranial pressure and the coma it produces.

The same fact may also explain another difficulty, for looking upon uræmia as organic uric-acidæmia, because its symptoms all resemble so closely those of functional uric acidæmia (see "Uric Acid," pp. 226-7), it was a surprise to me on examining some blood drawn by cupping glass from a patient who was dying of uræmia, to find little or no uric acid. The patient, however, had considerable fever at the time the blood was drawn, and fever, as we know ("Uric Acid," pp. 57 and 118), clears the urate out of the blood and relaxes the arterioles. The fact, therefore, that I found no urate did not prove that it had not previously been there, and we know that there is often a large amount in the blood in Bright's disease, it being, as I have suggested, unable to pass freely through an inflamed kidney, and Grashey's suggestion about the effect of high tension on the cerebro-spinal fluid may explain why the cerebral symptoms did not depart along with the uric acid, when the temperature rose.

Just as the uric acid headache corresponds with a fluctuation in the excretion of urates, so very often do the fits of epilepsy, and the mental condition in each case fluctuates with the urates, there being mental brightness and well-being with the minus excretion which precedes the attack, and more or less misery and depression with the plus excretion that accompanies the attack; and so constant is this that quite a number of cases have been recorded in which the sufferers were able to predict an attack of headache or fits, as the case might be, from the fact that they were feeling more than usually bright and well.

Then, again, in many cases, the fits appear, if we may take the word of the patient, to be very decidedly influenced for good by drugs such as acids, opium, mercury, salts of zinc, copper, silver, nitro-glycerine, &c., which diminish the excretion of urates and clear them out of the blood.

And as to diet, very numerous cases are recorded in which a vegetarian diet has effected a cure, and there is, I think,

XUM

but little doubt of the way in which this diet acts in the parellel case of the uric acid headache.

I have now before me the notes of a case in which a gentleman, aged 50, who had suffered from fits all his life, was cured by a severely vegetarian diet, with a purgative dose of calomel and colocynth, followed by haust. Sennæ co. twice a week, and he lived twenty years more without any fits at all.

He lost weight very decidedly, and was considerably weakened by the treatment, and I think it probable that the urate formation was not only greatly reduced, but that the heart was also much weakened by the severe treatment, and hence was incapable of keeping up so much tension as was necessary to compress the intra-cranial veins.

Probably this treatment was unnecessarily severe; at the same time there are, no doubt, plenty of epileptics who would be glad to purchase freedom from their disease at even greater cost.

The treatment in the above case was recommended by a Dr. Pelham Warren, long since dead, and the directions say, among other things, "the medicine is meant to remedy the mischief arising from an inactive state of the liver, indigestion, &c., but the constitution cannot be freed from the unhappy tendency to pressure on the brain and convulsions without the strictest persevering attention to diet."

"Pressure on the brain" is an extremely good and possibly accurate description of the real condition present, and it reminds us of what patients with headache or depression so often say—that their head is bursting, or their brains boiling over. We can now realise much more thoroughly the condition of affairs that underlies these sensations.

As to epilepsy, some interesting researches have been published by Drs. C. A. Herter and E. E. Smith (*New York Medical Journal*, August, 1892.)

In the publication of some previous researches on uric acid, these authors were inclined to take objection to my results on account of the process used; but since that, I and others have pointed out that Haycraft's process is an ex-

XUM

tremely accurate one (see *Journal of Physiology*, vol. xiii.), and that as regards most of my results, some very similar, if not identical, ones have been obtained by others with various processes.

As regards epilepsy, I have pointed out that the excessive excretion of uric acid which accompanies the attack is sometimes so obvious as to be visible to the naked eye as a deposit falling through the urine before it is cool (see "Uric Acid," p. 87), and where the fit is well marked and the separation of urine accurate, an excess of uric acid can be found by almost any process.

As regards the remarks of Herter and Smith on epilepsy (prev. ref., p. 224), it is quite evident that in the case of grand mal they have not sufficiently separated the urine corresponding to the fit, and, as I have frequently pointed out before, it is quite useless to expect a positive result if this is not done.

The smallest amount of urine examined in their tables is 134 cc., and this was not at the time of an attack of grand mal. All the amounts estimated at the time of an attack of grand mal were larger than this.

Now the amount of urine excreted in an ordinary grand mal attack, lasting, including the stupor following the fit, 20 to 30 minutes, could not be much more than 30 to 40 cc.; therefore, even under the most favourable circumstances, the urine of an attack was mixed with two volumes of urine excreted before or after it, and no excess of uric acid would therefore be expected.

As I have pointed out, even in the case of the uric acid headache, the excretion of the whole day may show minus uric acid, while if the urine of the attack is carefully separated, it shows plus uric acid corresponding to the hours of headache (see *Journal of Physiology*, prev. ref.). In my case of epilepsy published in the *Neurologisches Centrallblatt*, to which the authors refer, the urine of each fit was separated as carefully as possible; and I also pointed out that when the urine of the fits was mixed with that before and after them, the uric acid came out normal, while the urine secreted during the fits showed a great excess.

In other cases I have got patients to pass water every hour of the waking day; then, when a fit occurred, we had the urine of the hour preceding the fit ready for examination, and the bladder never contained more than one hour's urine in addition to that of the fit, and this, in my opinion, is the way in which a uric acid reaction should be looked for in grand mal. Certainly it is quite useless to examine 150 to 500 cc. of urine when that excreted during the fit could not possibly exceed 20 or 30 cc.

The authors say again on p. 234: "The latter [that is, urines passed immediately after seizures] are apt to show a higher uric acid ratio than the urines passed just before seizures; very often, however, the ratio is one that belongs within the limits of health."

This is just my result diluted, and as the quantities are not given there is nothing to show that the urine just before was not the urine of two to three hours before, *i.e.*, a mixture; or that the urine passed after the fit really corresponded only to the excretion of the fit.

Further down on the same page they say: "The excess of uric acid that is observed in epilepsy cannot reasonably be construed as the cause of the seizures." They acknowledge, then, that there is an excess, but differ from me as to the relation of cause and effect.

In petit mal, however, they found a distinct relation between the excretion of uric acid and the cause of the seizures, and treatment by milk diet greatly reduced the frequency of the attacks (prev. ref., p. 239.)

This is just what I should expect, as in my writings on the subject I have not limited myself to grand mal, but have suggested that petit mal, hystero-epilepsy, and hysteria are all similarly related to uric acid (see "Uric Acid," p. 87 and 123).

Where attacks of petit mal are frequent I should expect a continuous plus excretion of urates; but, as I have often proved, it is useless to look for urate reaction in a single short attack, as the separation of urine can never be sufficiently good (prev. ref., p. 87).

If Herter and Smith will separate the urine of grand mal

cases with greater precision, I have no doubt that they will get urate reactions similar to mine, and if they will also treat grand mal to the same diet as petit mal they will, no doubt, get some satisfactory results, just as others have already done.

The observations of these authors on the relationship of the fits to intestinal putrefaction are of extreme interest because they supply a further illustration of a point which I had previously noticed. Thus, in St. Bartholomew's Hospital Reports, vol. xxiv., p. 226, I have made the following remark: "Salicylates again may fail to be absorbed for a time from gastric irritation or other causes, and I had a remarkable instance of this in a case of epilepsy which I was treating with this drug. On several occasions the urine gave no reaction with perchloride of iron during the fits, though on some occasions there was probably as much as 20grs. of the salicylate lying in the stomach, and there was a strong reaction in the urine after the fits had passed off, without any more of the drug being given."

I can now explain all the facts much more completely than I could at the time the above was written, for I now know that when uric acid in the blood raises arterial tension it contracts the arterioles and capillaries throughout the whole body; so that not only are the urine and saliva scanty, not only is the skin cold and dry, but all the gastro-intestinal secretions (gastric juice, bile, pancreatic and intestinal juices), are similarly diminished, and the circulation through their mucous membranes is so imperfect that digestion and absorption come to a standstill. This and the generally diminished metabolism accounts for the marked fall in urea excretion that generally accompanies a plus excretion of uric acid, and the resumption of these physiological processes accounts for the rise of urea when the urates are cleared out of the blood, and their excretion in the urine diminished.

But such a marked arrest of gastro-intestinal digestion will almost certainly be accompanied by the onset of putrefactive processes—the processes of which Herter and Smith have observed some of the results.

All is now clear; the reason the salicylate failed to appear

XUM

in the urine in the above case was because the arterioles were contracted, and gastro-intestinal digestion and absorption were at a standstill.

We can now see clearly the origin of the horrible fœtid dejecta often met with after an attack of dyspepsia, bilious attack, or uric acid storm; also why calomel so promptly puts matters of this kind right. I used to think that it acted directly as an antiseptic, but it is now evident that its most important action is the result of its chemical relations to uric acid, by which it clears urates out of the blood, relaxes the arterioles and capillaries, and allows gastro-intestinal digestion to proceed once more.

The intestinal putrefaction noticed by Herter and Smith, far from being the cause of the fits, is but a result of uric-acidæmia. The contraction of arterioles which produces the high tension, which in turn interferes, as I have pointed out above, with the intra-cranial circulation and produces the fits, interferes also with gastro-intestinal digestion and allows putrefaction to take its place.

Mental depression, again, is influenced by diet and drugs in just the same way as the above troubles; there are plenty of recorded cases in which a vegetarian diet has done good or effected a cure.

Hysteria is another instance of the same thing; its attacks bear the same relation to the excretion of urates as do those of the above-mentioned troubles, and, like these, it can be relieved by acids and other drugs which clear urates out of the blood; and I have suggested that the fluctuations in the excretion of urea that accompany and follow such attacks[1] represent the effects of uric acidæmia on the metabolism, for uric acidæmia, however produced, is accompanied by a diminution in the excretion of urea, which I have suggested is due to a diminished metabolism, the result of the general vascular contraction which the uric-acidæmia produces; and when the uric-acidæmia is put an end to, whether by drugs or by natural causes, and whether it has produced headache, mental depression, hysteria, or fits, the relaxation

[1] See Researches of MM. Gilles de la Tourette and Cathlineau in the *Progrès Medical*, vol. i., 1889.

of arterioles is quickly followed by a rise in the output of urea (see "Uric Acid," p. 122-4).

We can now see that the relation of hysteria to the uterine functions, and especially to menstruation, is due to the fact that this, even in physiological conditions, is almost certain to bring about a fluctuation in the excretion of urates, and a temporary uric-acidæmia, which, in turn, may produce hysteria, or any other members of the group, by its effects on the intra-cranial circulation. (See BRAIN, 1891.)

These considerations seem to me to warrant the conclusion that in none of these so-called neuroses does the disturbance of function ever originate in the nervous system, which would work on with the utmost regularity if its circulation was not disturbed.

But we can also see quite clearly that intra-cranial lesions, whether traumatic, syphilitic, or of other origin, may have a powerful influence in hindering or increasing the effects of uric acidæmia on the intra-cranial circulation.

As I have said before with regard to headache, it is quite unnecessary to treat the nervous system at all if the uric acidæmia is prevented, and it appears to me to be quite evident that many of those who have believed that they were treating the nervous system direct, have really been treating the urid acidæmia unknown to themselves; for such drugs as morphine, the nitrites, the iodides, the salts of zinc, copper, silver, and mercury, have a powerful effect on the excretion of urates, and tend to remove uric-acidæmia and its effects on the intra-cranial circulation; and even the bromides with their depressant action on the muscles and the heart, may, like tobacco (mentioned before), affect the intra-cranial circulation rather than the tissues of the brain direct.

It is evident, then, that we can not only completely account for the relation which these functional disturbances have been found to bear to a large excretion of urates, and can see that this depends on their both being the results of one common cause—an excess of urate in the blood; but the explanation places us in a more favourable position than we have ever previously attained for making an attack on the real *fons et origo mali*.

In conclusion, I will shortly mention one or two points in treatment.

It is impossible to prevent fluctuations in the excretion of uric acid; they occur in every one from hour to hour; therefore the only way to prevent severe uric acidæmia is to diminish the formation of uric acid.

A wave 10 feet high at low tide may do little or no damage, while a wave of similar height at high tide may sweep everything before it. We must keep the uric acid tide always low, and then its waves will do no damage.

It appears that uric acid is generally formed at or about the proportion of one grain for every 33 grains of urea; therefore we can take urea as our guide, and, reducing its formation, can calculate that of uric acid from it.

If a patient suffers from headache or mental depression, and passes on ordinary diet 600 grains of urea per day (*i.e.*, forms 18 grains of uric acid), let him try the effect of such diet as will reduce his urea and keep it about 400 grains per day (*i.e.*, forming 12 grains of uric acid), and such a diminished output of urea may be effected without either starvation or loss of weight.

It seems also to be advisable to avoid meat which contains much urate already formed, also soup, beef-tea, and meat extracts, which are, as I have shown, very rich in urates (see "Uric Acid," p. 51).

In epilepsy it may be necessary to go further, to reduce urea still more, and even to produce considerable loss of body weight, as in the case I have mentioned.

I have great hopes that by careful advance in the direction which these facts indicate, we may eventually obtain a really valuable power of control over these functional disturbances.

XUM

[259]

Critical Digest.

ON RECENT APPLICATIONS OF GOLGI'S METHOD TO THE STUDY OF THE NERVOUS SYSTEM.[1]

BY WILLIAM ALDREN TURNER, M.B., M.R.C.P.

THE examination of the minute anatomy of the central nervous system by Golgi's staining method, which has brought forth much valuable information at the hands of Golgi, Kölliker, Ramón y Cajal and others on the Continent, has as yet received but scant attention in this country. It is therefore proposed to give here a *resumé* of the more important papers which have appeared on this subject, the literature on which is now voluminous.

The *method* of staining, although modified in details by various workers, is briefly as follows :—Small portions of the central nervous system of embryos are placed in a mixture composed of bichromate of potash (4 parts) and perosmic acid (1 part). After remaining in this solution for several days, the blocks of tissue are washed in a quarter per cent. solution of nitrate of silver and finally placed for two days in a three-quarter per cent. solution of this salt. They are then ready for imbedding either in paraffin or celloidin. However carefully done, the method has its disadvantages, which are, that all the nerve structures are not equally and at the same time stained, so that the relation of the different elements, *e.g.*, nerve fibres and nerve cells, is not always evident.

Kölliker finds that the nerve fibres and their branches stain most readily, so that one sometimes sees sections in which, to all appearance, there are no nerve or glia cells.

The *theory* of the action of this staining method has provoked discussion. It is held by some that the black staining which is characteristic of it is due to the precipitation of bichromate of silver in the pericellular lymphatic vessels; the reasons for this statement being that the cells are larger than where observed by

[1] This has already appeared in a slightly modified form as a series of papers in the *Journal of Anatomy and Physiology*, vols. xxv. and xxvi.

XUM

other methods, and that their shape is different; that ammonia dissolves the deposit revealing a smaller cell, and that the lymph channels in other organs are also stained by Golgi's method. This view is not accepted by all in its entirety, for Kölliker believes that both a staining and a precipitation takes place. If the elements present a sharply-defined smooth contour, it is indicative of staining, if they are irregular and varicose, precipitation has occurred; the former is chiefly observed in non-medullated fibres, the latter in neuroglia cells. Ramón y Cajal inclines to the view that the reaction is due to staining and not to precipitation: he has never obtained deposits in the lymphatic spaces of the cornea, lymphatic vessels or conjunctival lacunæ, while he has constantly seen the cells in epithelium, cartilage, and muscle stained by this method.

This preliminary introduction upon the method is now followed by a consideration of the anatomy of the central nervous system, which has been obtained mainly from the works of Golgi, Ramón y Cajal, Kölliker, and Van Gehuchten. A bibliography at the end summarises briefly the important works on the subject.

A. Spinal Cord (Kölliker).

1. *The posterior roots and posterior columns.*—The posterior nerve roots enter the cord by numerous small bundles which gently ascend in the postero-lateral groove; and turning mesially towards the posterior columns, each fibre divides into two portions, an ascending and a descending. In men and pigs this division takes place at angles of 150° or 160°, and as the fibres of a root bundle split at the same level, and the divisions retain their oblique course for a long time, the superficial portions of the posterior column present a peculiar and characteristic arrangement of fibres. In their further course these fibres extend into the deeper parts of the column and become longitudinal fibres. When examined more closely, the posterior roots are found to sink into the region of the lateral segment of the substantia gelatinosa in the part of the posterior column recently called the border tract (Randzone) of Lissauer. Here the root bundles divide into two systems, a smaller lateral one, which passes towards the postero-lateral corner of the substantia gelatinosa, and a stronger, stouter median one, which courses in the border tract towards the posterior column. In the lateral system all the root fibres divide at the same place; in the median they diffuse over the whole space between the entrance of the roots and the posterior column, occupying in antero-posterior diameter the ventral

part of the border tract, while the dorsal portion is formed by a border of longitudinal fibres. The divisions are here scattered over a considerable area; and the ascending and descending fibres which are given off from these so intertwine amongst each other, that in cross sections a very delicate alternation of small bundles of fibres exists. As a result of his investigations on the spinal cords of human embryos, as well as on those of sheep and newborn kittens, Kölliker is inclined to believe that all the sensory root fibres divide.

It is important to consider now the further course of the branches of the "sensory" roots, and to find out definitely how they end. The posterior columns, by which are meant not only the usual so-called columns, but also the connected layers of white matter which are situated outside the substantia gelatinosa and connect the posterior and lateral columns, are essentially composed of the fibres of the posterior roots.[1] The longitudinal fibres of the posterior columns bend at a right angle and pass into the substantia gelatinosa, where they break up into fine branches (in the spongy substance), and behave in a manner similar to what is later on described as occurring in the "collaterals" of the column fibres. These bendings are not abundant all over, and it is impossible to say whether they are only in the descending or also in the ascending fibres. Some are true terminations, and divide in the posterior horn into delicate branches, others are longitudinal fibres which turn back in the substantia gelatinosa by another bending to their former course. It is difficult to say how many of the branches of the posterior root fibres pass into the grey substance to end there, and how many pass up to the medulla oblongata. It is most probable that all the descending branches pass into the grey substance, and that the ascending mostly pass up to the brain. Kölliker believes also that these latter may partly enter the grey substance, as he has observed in longitudinal sections that some of the fibres entered the grey matter at right angles and ended there. Observation of many sections further shows that a great number of the fibres of the posterior columns run a very considerable distance without interruption.

It is of interest to notice if this method throws any light upon the vexed question of the origin of the "sensory" roots in cells of the grey matter of the cord. The observations of Golgi, Ramón y

[1] It is essential to remember that the posterior horn and substantia gelatinosa is of extraordinary breadth and thickness in the embryo. As growth proceeds, the grey matter, which at first prevailed, gradually gives place to the white.

XUM

Cajal, and Kölliker cannot confirm this statement; none of them has ever seen a nerve cell passing into a "sensory root" fibre. Until further proof is forthcoming for the higher animals, it is well to teach that the origin of the "sensory" root fibres in the cord cannot be held as proved.

2. *The Collaterals (Collateralen) of the Posterior Roots and Posterior Columns.*—The existence of lateral branches (collaterals) of the longitudinal fibres of all the columns of the spinal cord is a most important discovery of Golgi and Ramón y Cajal. They are very readily seen in longitudinal sections, and are a noteworthy feature of the minute structure of the cord, and form one of its most important physiological relations. As a rule they are not so thick as the longitudinal fibres, and like them they present varicosities, especially on their fine ramifications and at their terminations, which, according to Kölliker, are artificial. The collaterals are usually given off at acute angles, but in some cases they arise from the stem fibre at a right angle.

All collaterals end in small *terminal bushes or tufts*, which remind one of simple end plates. These small tufts consist of numerous short twigs of the finest varicose fibrils, which encircle the bodies of nerve cells, and mainly end in fine knobs. When the cells are large, as in the anterior and lateral horns, these twigs form very beautiful pictures, and one notices that there are no anastomoses of neighbouring twigs, and that they are not in direct association with the cells. Similar endings are found in the substantia gelatinosa, substantia spongiosa, and in Clarke's column. As at their terminations the collaterals show no sign of anastomosis in their course.

We will now consider in detail the collaterals of the "sensory" roots and their branches and of the posterior columns. After these roots have entered the border tract, they give off in certain cases before their division single lateral branches. Similarly the fibres of the border tract and posterior column give off collaterals. The number of collaterals given off is very great, varying from two or three up to nine in very long fibres, and they follow each other at greater or less distance. They occur in all parts of the cord, both in the limb enlargements, in the intervening portions, and in the upper cervical region, and so many are there that it is difficult to see a fibre which does not give them off. In all portions of the posterior columns, superficial, middle, and deep collaterals are present; it is therefore reasonable to conclude that the majority of the fibres of the posterior column give off collateral branches. It is, however, worthy of note that in many preparations the col-

laterals are less well or not at all stained. In the dorsal corner of the posterior column of the cervical cord of a calf embryo, Kölliker noticed a small compact bundle which possessed no collateral fibres, while they were well formed in all the other portions of the column.

The sensory collaterals, as the lateral branches of the posterior column fibres may be called, pass from the different portions of the column into the grey matter of the posterior horn, and course more or less towards its ventral or motor parts, in order to terminate in its different regions, and even in the anterior horn of the same side. Their passage into the grey matter is in bundles of five, ten, fifteen, or more fibres, which enter partly in a straight course, and partly arched and slanting through the substantia gelatinosa. The place and nature of their termination is important—

(1) In the substantia gelatinosa in the manner described above as characteristic of collaterals.

(2) In the boundary layer between the substantia gelatinosa and spongiosa; here the fibres are numerous, and form a delicate felted arrangement.

(3) In Clarke's column, where they form a network similar to that in 2.

(4) In the substantia gelatinosa of the opposite side. In some young animals a posterior commissure is found to exist and to be composed of collaterals. Ramón y Cajal describes the posterior commissure in new-born dogs as consisting of three parts: anterior and posterior arching bundles, and a middle transverse portion. The anterior bundle has its concavity towards the central canal, and consists of delicate and coarse fibres, which spread out laterally in all directions. The origin of these fibres is doubtful. The posterior bundle has its convexity to the ventral side, and consists of the collaterals of the posterior columns, which end in the opposite side in the adjoining portions of the substantia gelatinosa. The middle (a cross-running bundle) goes midway through Clarke's column, and ends in the lateral part of the substantia gelatinosa. The elements of this part of the commissure arise partly in the collaterals of the lateral columns, and partly their origin is doubtful.

(5) In the substantia spongiosa of the posterior horn of the same side.

(6) In the anterior horn. There is a very important group of the sensory collaterals which penetrate the anterior horn of the same side, called by Ramón y Cajal the "antero-posterior" or

"sensori-motor" group. Springing partly in the posterior column and partly in the border tract, they collect in the substantia spongiosa into stout bundles, and penetrate as compact strong fibres straight into the anterior horn, where they spread to all parts, and end in the region of the nerve-cell groups in the finest branches. These are defined by Kölliker as the reflex collaterals of the sensory roots.

3. *Collaterals of the Anterior and Lateral Columns.*—The collaterals of the lateral column fibres pass partly into the ventral portion of the posterior column and of Clarke's column, and partly into the anterior horn to the various motor cell groups. Some single fibres pass into the boundary region between the substantia gelatinosa and spongiosa and towards the white commissure. The collaterals of the anterior columns all pass dorsalwards, some cross into the commissure, others towards the anterior and lateral horns of the same side, while others still go into the posterior columns. These latter form end bushes, which remind one of the long sensory collaterals, and are most perfectly recognised in sagittal sections. Concerning the end ramifications of these collaterals, Kölliker found, what he had not previously noticed in the sensory ones, long dependent branchlets, which give off on both sides, at nearly a right angle, a great number of short branches, which end as above described. Many of these collaterals only show such divisions, but others are in all respects similar to the sensory ones.

The anterior commissure consists partly of axis cylinders, which decussate, and partly of collaterals of the anterior and lateral columns, which often show branchings in the commissure and end in the grey matter of the opposite side, where they partly spread out into the anterior horn and partly turn toward the posterior horn. It is not impossible that among the last-mentioned fibres posterior column collaterals exist, which pass through the commissure to the opposite side.

4. *Nerve Cells.*—The cells of the grey matter of the spinal cord are all multipolar, but differ essentially, in that (1) some possess one unbranched or only sparsely-branched nerve process, (2) while in others this gives off numerous branches; this latter set are again divided into two sub-classes: (*a*) those in which the axis-cylinder process, in spite of its giving off branches, does not lose its individuality; and (*b*) those whose axis-cylinder process divides into delicate and very fine ramifications. According to Golgi's classification, the cells of the first category and of (2) (*a*) are motor, while those of (2) (*b*) are sensory.

Another classification of nerve cells is based on physiological grounds, and is as follows:[1]—(*a*) *Motor Cells*, which pass into the motor root fibres or give origin to the same. These cells lie in the anterior horns and form nerve-cell groups; they are as a rule large, but may be small, especially in the median part of the ventral segment of the anterior horn, and in the region between the anterior and posterior horns. The nerve process of these cells goes usually straight towards the exit places of the roots, but others are arched. It is most probable that these fibres are not branched, although Ramón y Cajal mentions that he once saw in a rat a lateral branch of such a fibre coursing backwards. Golgi also describes such. It is yet doubtful whether these are constant or only in single cases. This point is of great importance physiologically, and the simplest explanation of such a branch is that a motor cell may in certain cases branch out into centrifugal fibres.

(*b*) Cells whose nerve process is transformed into a longitudinal fibre of the white matter [Strang-zellen—column fibre cells]. They can be sub-divided into (1) cells whose process remains on the same side of the cord, (2) cells whose process passes by the white commissure on to the opposite side [Kommissurenzellen of Ramón y Cajal]. The great majority of these cells send their nerve processes into the lateral columns, many also into the anterior; the former lie mainly between the central canal and the projecting median corner of the lateral column; the latter are especially related to the median cell group of the anterior horns. The nerve processes of these cells partly possess no branches; in many cases, however, they give off a great number of branches without losing their individuality. The manner of ending of the stem fibre of the nerve process is noteworthy. In the majority of cases, as Golgi first saw, the end of a stem fibre bends nearly at a right angle into a column fibre. Kölliker considers all as nerve fibres which bend out of the longitudinal columns into the grey matter at a right angle, especially if they show no diminution in their diameter, and if they cannot be followed up to a nerve cell. Secondly, the stem fibre ends by dividing, forkwise, into ascending and descending column fibres, just as the sensory roots do at their entrance. And, lastly, there remain a few processes, whose relation in the white substance is not yet settled, whether they pass away in one or in divers directions.

[1] The examination of the axis-cylinder processes of the cells of the cord is one of very great difficulty, owing to the fact that by the Golgi method, as already pointed out, all the cells are rarely stained.

According to Golgi and Ramón y Cajal, all the nerve processes of this category pass into column fibres. In a preliminary communication Kölliker believed that those which crossed in the anterior commissure passed into the anterior roots of the opposite side. As he has been unable to find a relation between the commissure fibres and the nerve roots, he has no reason to hold his opinion any longer.

(*c*) Cells whose nerve processes give off branches right up to their terminations, but do not pass beyond the grey matter. These were first described by Golgi, and appear to exist only in the posterior horns, but not exclusively. They are most marked in the neighbourhood of the point of the posterior horn, but have also been found at the dorsal border of the substantia gelatinosa and in other parts.

Especially important for the interpretation of these cells was the nature of the termination of their branches. Golgi and Ramón y Cajal noted that they ended free and with varicose branches; none of them end in tufts or bushes, as is so characteristic of collaterals.

For accounts and figures of the manifold forms of nerve cells and of their protoplasmic processes, the reader is referred to the works of Ramón y Cajal. It is necessary, however, to say that it is very difficult to define the protoplasmic processes of these cells. When one remembers that these branches are perfectly smooth, the pointed and granular appearance which they present, and which makes them resemble neuroglia cells, is probably artificial. Regarding these processes the following may be noted:—in silver preparations they form numerous coarse ramifications, so that in the organs of the new-born many cross through the whole breadth of the grey matter, and occupy half of its antero-posterior diameter. The median group of cells of the anterior horn are peculiar in that they send a portion of their branches through the anterior commissure to the opposite side—a relation which Ramón y Cajal first pointed out, and which Kölliker finds also in the cells in the neighbourhood of the posterior commissure. Some protoplasmic processes enter the white columns, and often penetrate right through to the surface of the cord.

5. *Neuroglia Cells.*—These consist of the elements of the original medullary plate, and are divided into primary and secondary groups. The first appear as the epithelium of the central canal, and originally pass through the whole cord, but later on are only present in the deeper parts; the latter arise later in all parts of the grey and white substance, likewise from the elements of the medullary plate.

XUM

Summary.—1. The sensory root fibres divide at their entrance into the cord into an ascending and descending branch.

2. Some of these sensory fibres course for a considerable distance (4-6 cm.) longitudinally; others arch into the grey matter, and end free in delicate ramifications.

3. An association between the sensory root fibres and the cells of the grey matter of the cord has not up to the present been observed.

4. All sensory longitudinal column fibres give off delicate lateral branches—the so-called *Collaterals* of Ramón y Cajal, which, entering the grey substance, ramify in all its parts and end free.

5. The motor root fibres arise from greater and smaller nerve cells of the anterior horn in a single nerve process, which in certain cases (always, according to Golgi), give off lateral branches.

6. The anterior and lateral columns consist partly of fibres, which are given off by nerve cells in all parts of the cord. These column fibre cells (Strang-zellen) give off from their axis-cylinder process a greater or less number of lateral branches into the grey substance, which end free.

7. The relations of the nerve processes of the anterior and lateral column cells to the fibres in the white substance varies. In some cases they simply bend upwards and pass into a column fibre; at other times they divide into two or three branches; or, again, such a nerve process splits into an ascending and a descending fibre, or apparently attaches itself laterally to a column fibre (the T-shaped fibres).

8. The great majority of the fibres of these columns give off collaterals, which enter the grey matter of the anterior horn and of the anterior part of the posterior horn, and there end free, just as the sensory collaterals do.

9. The anterior and lateral column fibres bend in many cases at a right angle into the grey substance, and there end free.

10. All collaterals of column fibres, and all lateral branches of axis-cylinder processes end in the same way. They give off at acute or right angles a greater or less number of branches, and finally form delicate end bushes, which encircle nerve cells without associating themselves with them on forming any anastomoses.

11. The anterior commissure consists of:—

a. Axis-cylinder processes of nerve cells of the grey matter, which, after decussating, are continued as longitudinal fibres of the anterior and antero-lateral columns.

b. The decussating collaterals of the anterior and lateral columns.

c. The decussating protoplasmic processes of a part of the median cells of the anterior horn.

12. The posterior commissure consists of :—

a. The decussating collaterals of the sensory root fibres.

b. Possibly of the decussation of the collaterals of the posterior part of the lateral columns.

c. Decussations of the axis-cylinder processes of cells placed laterally to the central canal and substantia gelatinosa (Ramón y Cajal). But this is very doubtful.

d. Decussations of the protoplasmic processes of cells of the posterior horns. This also is not quite ascertained.

13. The nerve cells are divided into :—

a. Motor cells (*vide* under 5).

b. Column cells (*vide* under 6).

c. Cells, whose axis-cylinder process does not go beyond the grey matter, and forms delicate ramifications in this. These only exist in the posterior horn.

14. The value of the size, shape, and character of the nerve cells is not fully known. It is at any rate certain that large nerve cells are not limited to the motor sphere only.

15. The protoplasmic processes of all nerve cells ramify over a great extent in all directions, often penetrate the white matter, give origin to no nerve fibres, and do not anastomose.

B. The Cerebellum.

1. *Purkinje's Cells.*—Golgi showed the excessively rich ramifications of the so-called protoplasmic processes of these cells, and proved that they formed no anastomoses with each other. He believed that the most superficial of these branches were attached to the connective tissue at the margin of the molecular layer and to the vessel walls. He further made the very important discovery concerning the axis-cylinder processes of these cells, viz., that they gave off a certain number of lateral branches, which partly coursed back into the molecular layer and partly spread out into the granular layer.

2. *The Small Cells of the Grey Molecular Layer.*—Contrary to the statements of many authors, this method definitely showed that these cells existed throughout the whole thickness of this layer in not inconsiderable numbers, and possessed, besides

anastomosing branches, an axis-cylinder process which formed very delicate ramifications and coursed in very diverse directions, amongst which the horizontal was especially conspicuous. The end of this process was not seen.

3. *The Small Cells of the Granular Layer.*—Golgi first definitely pointed out that these were nerve cells. Their axis-cylinder processes are very slender, still lateral branches are occasionally found, and anastomoses of these processes with nerve fibres. The protoplasmic processes only divide very sparely, are short, and appear to end in small granule heaps (Körnerhaufen).

4. *The Large Cells of the Granular Layer.*—In human beings these exist as spindle-shaped bodies, and are characterised by the extraordinarily numerous ramifications of their axis-cylinder processes.

5. With regard to the *medullated nerve fibres*, Golgi showed that many ramified in the white matter, but especially in the granular and molecular layers, so that their very terminations showed a thick plaiting, the details of which it is impossible to follow out. These anastomose with—(1) The axis-cylinder processes of the small cells of the granular layer; (2) the lateral branches of those of Purkinje's cells; (3) the nerve processes of the small cells of the molecular layer. As the most characteristic part of this plaiting Golgi portrays those parts which lie at the boundary of the granular and molecular layers, and which present many coarse and fine horizontal elements.

Next to those of Golgi come the important observations of Ramón y Cajal, who used the rapid hardening method with bichromate of potash, osmic acid, and silver. The most important of these observations are briefly as follows:—

1. *The Small Nerve Cells of the Stratum Granulosum.*—The branched processes of these cells end in small tufts of closely-set short branches; the axis-cylinder processes ascend, without exception, into the molecular layer and form in this delicate longitudinal elements, running parallel to the convolutions. In this layer they divide at different levels at right angles into two longitudinal fibrils, .2 to .5 micromillimetre long, which after a course of .2 to 1 millimetre end free without branching.

2. *Small Nerve Cells of the Molecular Layer.*—The most note-worthy facts discovered by Ramón y Cajal upon these elements are that the axis-cylinders of all the cells lying near the granular layer form peculiar branches or baskets which encircle the bodies of Purkinje's cells and form holes for them.

3. In the cerebellar cortex he found also specially branching fibres, which passed from the granular into the molecular layer and which he divided into three kinds :—

(*a*) Fibres which passed from the granular into the molecular layer and ended there in a branched expansion. He was able to follow these fibres in some cases into the white matter.

(*b*) Fibres which, arising in the white substance, ramified in the granular layer, and showed in their course mossy appendages. These are the "Fibras musgosas" of Ramón y Cajal. The very terminations of these fibres, each of which possesses from 20 to 30 branches, are on the level of Purkinje's cells, and appear to pass over into longitudinal fibrils. He is inclined to construe these fibres as the sensory terminations of the axis-cylinders of peripheral nerves, and bases this hypothesis on these two facts :— (1) The optic nerve in birds ends in the lobus opticus in free branches ; and (2) the sensory root fibres in the cord course in a similar way.

(*c*) Fibres which, ascending from the granular layer, end in nests which encircle Purkinje's cells. These are the "Nidos cerebellosos."

4. Concerning the cells of the cerebellum described by Ramón y Cajal, in addition to those facts mentioned above, are the following :—

a. The axis-cylinder processes of Purkinje's cells have processes which pass back into the molecular layer.

b. The axis-cylinder process of the large cells of the granular layer is extraordinarily richly branched.

5. Ramón y Cajal mentions no branches in connection with the medullated fibres of the cerebellum. Concerning the question which of the fibres (which are shown by Golgi's method) are medullated, he surmises that possibly the very terminations of the "fibras musgosas" may be, and also the branches of the axis-cylinder process of Purjinke's cells which bend into the molecular layer, for both of these sets may become the medullated longitudinal fibrils of the molecular layer. As non-medullated, Ramón y Cajal considers the axis-cylinders of the granular cells, those of the small cells of the molecular layer, and of the larger granular cells.

Since the recent observations of Ramón y Cajal upon the cerebellum have not yet been confirmed, and even those of Golgi have only been re-examined by a very few, it will not appear superfluous if I communicate the results of Kölliker's own observations upon the minute structure of the cerebellum.

1. *The Small Nerve Cells of the Granular or Rust-coloured Layer, or the Small Granular Cells* (Körnerzellen).—The examination of these cells has only been made in cats, and in preparations which have been treated both by Golgi's long method and by the short bichromate of potash, osmium, and silver method. In both forms of preparations, but especially in the first, these nerve cells were found, as Ramón y Cajal described, to possess short protoplasmic and long axis-cylinder processes. The former averaged from 10-40 micromillimetres in length, were simple, and appeared to end in short branches; the latter sprang usually from one of the other processes, only seldom from the body of the cell, and penetrated with a serpentine course outwards into the molecular layer. In such preparations only single small granular cells are stained, so that they are easily followed in their entirety; yet one finds places at which two, three, or more cell-bodies are grouped together, and from which a great number of typical nerve and ramifying branches go off. It is not difficult to follow the fragile nerve-processes right into the molecular layer, and to prove that they are here transformed into longitudinal elements after they have divided into two branches, each of which forms a right angle with the stem. These longitudinal fibrils here and there apparently end free; but it ought to be emphasised that the free endings are possibly not such, for it is impossible to say whether the silver has stained them throughout their whole length. The number of these fibrils is very considerable, and longitudinal, oblique, and tangential sections of the convolutions show them in the form of a very thick and delicate striation of fibrils, some of which are varicose and tortuous, and pass through the whole thickness of the molecular layer to its most superficial parts. On cross sections of the convolutions these elements appear as fine dots or streaks, which fill in the intervals between the ramifications of Purkinje's cells.

In exceptional instances this longitudinal fibre-system has special relations. Kölliker agrees with Ramón y Cajal, firstly, upon the presence of branches to the oblique stem fibres within the molecular layer, which become longitudinal fibrils; and, secondly, upon the appearance of ostensible stem-fibres, which pass over at a right angle into only one longitudinal fibre. Ramón y Cajal thinks that such a fibre could represent the termination of a longitudinal fibre, and, since he never saw it in association with a small granular cell, but followed it well up to the white matter, could pass into a medullated fibre.

The following facts may be added regarding these longi-

tudinal fibres:—They stain most readily in the innermost parts of the molecular layer, and seldom throughout its whole thickness. Further, the staining is only partial, so that, between large and small bundles, streaks exist in which none of the fibrils are stained.

2. *The Large Multipolar Cells of the Rust-coloured Layer.*—These cells which have been discovered by Golgi have been confirmed by Ramón y Cajal. Their nerve processes form very rich ramifications. Kölliker saw them in cats and in the embryos of pigs and puppies, and, in contrast to those just described, he calls them the large granular cells. They are far smaller than Purkinje's cells, and lie either at the outer margin of the granular layer, partly almost on a level with Purkinje's cells, and partly in the middle of the layer, or even at the inner boundary up against the white matter. From an angular cell body there pass towards different parts three up to six or more branching processes, which course partly between the granules and partly pass into the molecular layer, and can often be followed far outwards in this layer; but besides they can penetrate into the white matter, as was once observed in the deeper parts of the cerebellum.

3. *The Nerve Cells of the Molecular Layer.*—The nerve cells of this layer are divided into two sets, the large and the small, of which the former are situated in the deeper and the latter in its superficial parts. While this last possess the usual form of multipolar nerve cells, the former present a very strange disposition of nerve processes, which, as Golgi and Ramón y Cajal pointed out, course transversely for a certain distance in the region between the molecular and granular layers. This transverse fibre gives off, at right angles, processes which encircle as in a basket the bodies of Purkinje's cells. The bodies of the cells measure from 20 to 25 micromillimetres, and are usually drawn out longwise, and present four, five, or more angles. Besides the nerve process, they give off a large number of protoplasmic processes, which, mainly passing outwards, ramify in the usual way, and extend as far as the outermost parts of the molecular layer. The so-called nerve process of Ramón y Cajal is an altogether peculiar structure. Beginning very delicately at the body of the cell, it becomes in its horizontal and transverse course twice or four times thicker, and stretches over the bodies of Purkinje's cells in order to give off branches from place to place. These branches pass off obliquely, sometimes with a coarse, sometimes with a delicate root, and end in such a way that each branch or branchlet is transformed into quite a tuft or pencil of terminal fibres, which

encircle basketwise the bodies of Purkinje's cells.[1] To unravel them further is a work of considerable difficulty, but it is probable that the branches have no association with each other, but end free. Likewise their relation to Purkinje's cells is not easy to ascertain. From transverse and longitudinal sections both parts appear to be only in juxtaposition, so that the baskets not only always closely embrace the cell bodies, but penetrate with single processes into the granular layer. It is important further to note that the pencil-like arrangement of branches of many cells participate in the formation of one basket. Kölliker confirms Ramón y Cajal's observations that a cell only possesses one process which lends itself to the formation of a fibre basket. Besides the descending branches which go to form the basket, the nerve processes of the cells in question give off here and there towards the surface of the molecular layer processes, which behave as ordinary protoplasmic processes.

The cells which participate in the formation of the above described fibre basket, and which for simplicity's sake should be called basket cells (Korbzellen), belong, as has already been remarked, to the deeper parts of the molecular layer. The number of these elements corresponds to the number of Purkinje's cells, and is therefore sparse in the region of the furrows. Still there may be other variations in the length of their processes and in the number of their pencils. Since one cannot follow one fibre from beginning to end, it is impossible to define its actual length. In the human species the basket cells measure 11 to 20 micromillimetres, the molecular layer .15 to .14 mm., and the part of the layer containing the transverse fibres .12 to .22 mm.

Following on the description of these remarkable basket cells, their branches, and the fibre basket, the question naturally arises if they are really nerve elements, as Golgi and Ramón y Cajal believe them to be, or glia cells.[2] This is no easy matter to decide, as by Golgi's method both neuroglia and nerve cells are stained by silver. Kölliker inclines to the view of Golgi, and states the following as matter of fact:—firstly, all transitions exist between the basket cells and the small undoubted nerve cells of the molecular layer; secondly, the glia cells have, without exception, numerous branches passing off from all sides, mainly with sharp-

[1] Kölliker has applied distinctive names to these structures. The basket or pencil arrangement of fibres he has called *Faserkörbe* and *Faserpinsel* respectively. The cells he has called basket cells (*Korbzellen*).

[2] In this connection it is to be remembered that Gierke speaks of the "Neuroglia holes" of Purkinje's cells, "Die Stutzsubstanz des Centralen Nervensystems," II. Theil., in *Archiv für Mikr. Anat.*, Bd. xxvi., p. 188.

angled divisions; and, thirdly, the basket cells are, like nerve cells, stained deep black, while glia cells have a red or yellowish-brown appearance. If the basket cells are really nerve cells, they exhibit structure of quite a peculiar nature, and the relation of branches of nerve cells to other such cells as exist here are nowhere else to be seen. This is surely no reason against this interpretation, since the recent examinations upon the construction of the retina and the brain show that we have to await much that is astonishing from further researches in this sphere.

4. *Purkinje's Cells.*—These are obtained readily in the human brain, and by Golgi's method present a picture of wonderful beauty. Their branches do not anastomose, and they pass out through the whole thickness of the molecular layer. Kölliker has not seen any connections between them and the pia mater or vessels, and only to a very small extent with the nerve processes of the small granular cells.

In relation to the position of Purkinje's cells, Henle first pointed out that their ramifications spread out in one single level, and were leaf-shaped, and that these leaves without exception lie in the cross plane of the convolutions. Deep tangential sections give good information upon the position of the bodies of the cells; these show that they are somewhat nearer together in the longitudinal than in the cross direction, and are not arranged in regular succession. Golgi has so well described the nerve processes of these cells, and Ramón y Cajal has confirmed it, that it is only necessary to add that Kölliker also saw the lateral branches which pass back into the molecular layer.

5. *The Nerve Fibres of the Granular and Molecular Layers.*—Of the three kinds of fibres which Ramón y Cajal describes as ramifying in the granular and molecular layers, Kölliker believes that the sub-sections (*a*) and (*b*) described above belong together, and correspond to the system of nerve-fibres which Golgi saw diffusing from the folia into the cortex. These varicose black fibres show inside the folia single acute-angled divisions, and so soon as they enter the granular layer they spread out into delicate rich ramifications, which pass through the granular layer and appear to end partly there and partly in the deeper portions of the molecular layer, *i.e.*, they end free, and are not further stained. In many preparations these endings show nothing special except slight varicosities, in others knotty or bushy swellings; it is probable that these conditions are artificial. As to Ramón y Cajal's third category, the so-called "Nidos Cerebellosos" which encircle Purkinje's cells, Kölliker only saw them

in a single preparation from a young dog, and is disinclined to pronounce any opinion on them.

6. *The Medullated Fibres of the Cerebellum.*—For the investigation of these fibres, we possess important means in the methods of Weigert and Pal.

At the borders and lateral parts of the convolutions, where the grey matter pushes against the white substance, single fibres and fibre bundles pass off from this in order to pass through the entire thickness of the rust-coloured layer, and to form in it a plexus, which takes up the granules in its meshes. In the deep parts of the furrows, different relations are found; here the white matter shows arching fibres, which, similar to those of the great brain, appear to act as commissural fibres of the convolutions, and only give off a few fibres to the rust layer. On examining this plexus more carefully, one finds that it contains a certain number of coarse fibres, which run straight towards the molecular layer, elements which undoubtedly are in great part continuations of the axis-cylinder process of Purkinje's cells. On the other hand, one cannot help thinking that the plexus of the rust layer consists entirely and alone of such fibres as Henle surmised. Still much more certain is it that it contains an excess of medullated fibres, which penetrate into the molecular layer. If the plexus is followed right up to Purkinje's cells, one finds that its elements pass under the cells into a layer of cross fibres, which, on cross section, consists of discrete bundles which are joined together by numerous fine bridges. Longitudinal sections complete the picture, and show the bundles as a not sharply-defined round collection of points. From these superficial cross bundles a quantity of oblique and straight ascending fine fibres develop, which, encircling Purkinje's cells and passing between them, enter the molecular layer, and in this strike into a longitudinal direction. In clean cross sections these elements appear as small points, which lie sometimes outside, sometimes between Purkinje's cells; and in longitudinal sections of the convolutions one finds a zone of fibrils running parallel to their surfaces. Besides these elements one finds a certain number of radial (straight and slightly oblique) ascending medullated fibrils in the molecular layer, of whose quantity it is not easy to make a calculation. They are, as a rule, few in pure cross sections, abundant on longitudinal sections of the convolutions. With reference to the diffusion of these medullated fibres throughout the molecular layer, recent examinations have given different results than previously were noted. There is no doubt that they

are more numerous in the inner fourth or fifth parts of this layer, but they are also observed in the superficial strata, just under the pia mater.[1] Do the medullated fibres of the cerebellum possess branches? It is extremely difficult to define such branches, except in the molecular layer. The reason of this difficulty is found in the fact that the medullated sheath is absent at the divisions, and therefore escapes staining. This has long been known to occur at the division places of peripheral nerve fibres. Ramón y Cajal and Flechsig think that this occurs in the central nervous system. The former has seen non-medullated portions in the medullated nerve processes of Purkinje's cells, while the latter has described non-medullated places in the already medullated nerve processes of the pyramidal cells of the cortex cerebri, where they divide and give off lateral branches. Kölliker has also seen such conditions in the fibres of the granular and molecular layers, but could not say whether they were natural or only imperfectly stained. In well-stained Weigert preparations from man and cats he saw such divisions in the molecular layer in only thirteen cases.

7. *The Neuroglia.*—In the white matter numerous large star-shaped cells are found, whose long-branched processes course between the nerve fibres, and apply themselves to the vessel walls. Similar cells are seen in the granular layer and at the border of the molecular layer, which send their long-branched processes right through this layer to its surface, to end at the inner aspect of the pia mater in a small club-shaped swelling.

C. The Cerebral Cortex.

Ramón y Cajal has examined the cortex in small mammals (rats, rabbits, &c.). He divides it into four layers. *First or molecular layer.*—This, the most superficial layer, is composed of nerve fibres, nerve cells, and the protoplasmic expansions of the subjacent pyramidal cells. The nerve fibres are extraordinarily abundant, and run parallel to the surface of the cortex; some of them have been observed to pass into the subjacent layers, and become continuous with axis-cylinder processes. Of the nerve cells three kinds have been described:—(*a*) Polygonal cells, not numerous, and possessing protoplasmic processes which pass off in all directions. (*b*) Fusiform cells; these he

[1] It seems probable that in Weigert preparations dark-stained glia fibres may be mistaken for very fine medullated nerve fibres. The former are of equal diameter and always serpentine; the latter are straight and usually slightly varicose.

found lying in a horizonital antero-posterior direction. They have only two protoplasmic processes, one from either pole. Owing to their great length it is almost impossible to follow out their whole course, but they bend and end free under the surface of the cortex. The axis-cylinder processes (for there may be two or three of them) arise from the protoplasmic expansions at a considerable distance from the body of the cell. They give off at right angles numbers of ascending collaterals, which end free in this layer. It is not uncommon to find a supernumerary axis-cylinder. The fusiform cells are not numerous. (*c*) Triangular cells; these are much more plentiful than the preceding; of the three branches which each cell gives off, one is descending and bifurcates. There are two, three, or more axis-cylinder prolongations, which always arise from the protoplasmic processes. Some pass to the surface of the layer, and others terminate free. The protoplasmic expansions of subjacent pyramidal cells ramify to a considerable extent in this layer, and end under the free surface. There are also oblique and arciform branches which end in different parts of this layer. Here, then, is an arrangement similar in all respects to that which obtains in the molecular layer of the cerebellum. In his previous works Ramón y Cajal had considered the protoplasmic processes not as absorbers of nutrition, but as the means of communication of nervous action between neighbouring cells and elements at a distance. In applying this principle to the molecular layer of the cerebral cortex, one observes that the fusiform and triangular elements, by means of their multiple axis-cylinder prolongations and very long protoplasmic expansions, are in contact with and intermix with a great number of the terminal protoplasmic processes of the subjacent pyramidal cells. It is not unlikely that some of these may functionate by contact. It is on this supposition that the term "association cells" has been given to the elements of the molecular layer.

Second layer (layer of small pyramidal cells).—In mammals these cells are not proper pyramids, but are polygonal or star-shaped. The protoplasmic processes pass off from the upper and lateral angles, and terminate in the molecular zone. The axis-cylinder descends, but its termination is yet the subject of much investigation. The author states that he has traced it into the white matter, and onwards into the corpus striatum (from cells in the frontal lobes), and into the corpus callosum (from cells in the Rolandic area). In their course they give off collaterals.

Third layer (layer of large pyramidal cells).—This layer passes insensibly out of the previous one. In mammals it resembles that

of man, except that the cell bodies are less pyramidal and more ovoid, and the protoplasmic processes are shorter. The axis-cylinder descends in a straight line into the white matter, where it becomes continuous with an association or projection fibre. It gives off many collateral branches, which assume a horizontal and oblique course, and end near the molecular layer. Nearly all the collaterals end in a small expansion without any branching.

Fourth layer (layer of polymorphic cells.)—The cells are mainly globular, but some are fusiform, and of various pyramidal types. They all have ascending and descending protoplasmic processes, the former of which end in the middle layer of the cortex, the latter in the white matter. The greater number of axis-cylinder expansions descend and give off fine collateral branches, which end as a delicate fibril in the white substance. This layer is in contact with the white fibres.

Scattered throughout these layers are certain other elements which require description:—1. *The sensory cells of Golgi.*—They are met with in all the layers, but mainly in the fourth or deepest zone. Their protoplasmic processes pass off in all directions, and are of considerable length, but they do not reach the molecular layer. The axis-cylinder descends, has a short course, and ends free in a complex ramification. Ramón y Cajal does not hesitate to disprove their sensory nature, as advocated by Golgi; he inclines to the view that they are "association cells." 2. *Cells with an ascending axis-cylinder.*—They are triangular, and lie amidst the small pyramidal cells. The axis-cylinder enters the molecular layer. Here it divides into horizontal fibres of great length. But all the cells are not prolonged so far as this layer; a similar disposition of branches takes place also in the second and third layers. 3. *White fibres which ramify in the grey matter.*—These are amongst the largest fibres which cross the cortex. They are larger than the axis-cylinder process of the large pyramidal cells. They penetrate the cortex at a right or obtuse angle, and divide into two or three large branches. These repeatedly divide, and end in the layers of small and large pyramidal cells. These fibres never anastomose with other fibres in the grey matter, nor do they terminate in cells. Ramón cannot confirm Golgi's belief that they are sensory. 4. *The nerve plexus of the grey matter.*—The intervals between the nerve cells are filled in by varicose fibres, formed by collaterals of the pyramidal and polymorphic cells, by the terminations of the sensory cells of Golgi, by the collaterals and terminals of the cells having an ascending axis-cylinder, by the collaterals of the association fibres of the white

matter, and by the collaterals and terminals of the fibres coming from distant central organs, and from the corpus callosum! The enormous complexity of the nerve network of the grey matter is sufficiently apparent from this description. No idea of the innumerable ramifying fibres in the cortex can be obtained by the Weigert-Pal method.

The white matter. 1. *Fibres proper to the cortex*, or association fibres.—The axis-cylinders of the cortical cells are continued into these fibres, either by simple bending at a right angle, or by dividing into a T or Y in the white matter; the branches of bifurcation pass in opposite directions. After some distance the fibres turn back into the cortex, and end by dividing. In the outer part of the hemispheres the fibres change their direction and form a large transverse descending tract—the fasciculus transversalis. When the fibres above mentioned divide into a T or Y, one of the branches goes to the corpus callosum. The association fibres give off collaterals, which pass at a right angle into the cortex, behaving just as the collaterals of the column fibres of the spinal cord. The presence of collaterals of the white fibres of the brain is now a well-known fact. 2. *The corpus callosum.*—The fibres of this structure have a horizontal course in the middle portion, but laterally they are ascending. They maintain their parallelism until they reach the subcortical white matter, where they bend amongst the arcuate fibres, and pass into the grey substance. They give off collaterals, which also pass into the cortex. It is not yet certain how these fibres commence or terminate. Some appear to be direct continuations of axis-cylinder processes; others are collaterals of the axis-cylinders of projection and association fibres. Their terminations are probably delicate ascending fibrillæ, which are found distributed through the middle and lower layers of the cortex. 3. *Projection fibres.*—These are the direct continuations of the pyramidal and polymorphic elements of the cortex. They give off a great number of collateral branches. They pass into the corpus striatum, where many are seen to give off very fine ramifying branches, which end in the cells of this ganglion.

Some general conclusions are worthy of note. One cannot tell by any special morphological character cells whose axis-cylinder constitutes either an association, a projection, or a commissural fibre. No direct anastomoses are observed between the nerve fibres of the grey matter. Transmission of nerve impulses must therefore be carried out through contact. The human cerebrum owes its superior activity not only to the great number of

its cells, but to the extraordinary richness of its association paths —*i.e.*, the collaterals, axis-cylinder, and protoplasmic expansions.

Van Gehuchten describes the *granular layer of the cerebellum* as composed of three kinds of cells—small and large nerve cells and neuroglia cells. Each small cell has a polygonal body, from each angle of which there passes a protoplasmic process, which ends freely in a tuft. The axis-cylinder rises from a protoplasmic process, and passes towards the molecular layer, where it bifurcates; its divisions end free. The large cells are met with in close relation to the cells of Purkinje. They have many protoplasmic processes, some of which pass to the surface of the cerebellum. Others are horizontal, and end in the granular layer. The *molecular layer* is rich in small cells. They give off protoplasmic processes, which pass upwards and downwards, while the axis-cylinder expansion assumes a horizontal course. Some of the collaterals of this pass to Purkinje's cells, which they envelop in a tuft, the so-called *Korbzellen* or basket cells of Kölliker. The protoplasmic processes of *Purkinje's cells* extend into the molecular layer; the axis-cylinder courses through the granular layer to the white matter.

Further study of the cerebellum has given rise to the same general conclusions regarding it as in both the cerebrum and the spinal cord. The true nerve element is the cell with its prolongations. The nerve elements are independent of each other, the protoplasmic processes end free, and, whatever its disposition, so does the axis-cylinder. Finally, the transmission of impressions here, as throughout the cerebro-spinal system, takes place not by continuity of structure, but by contiguity or contact.

Van Gehuchten has also paid attention to certain doubtful points in the spinal cords of small mammals. Firstly, the relation of the anterior nerve roots to the cells of the anterior horn. He finds that each anterior nerve root is the axis-cylinder prolongation of a cell. The protoplasmic processes of these "root cells" are numerous, and form an inner group, which passes by the anterior commissure to the opposite anterior horn; an antero-external group which passes to the periphery of the grey matter; and an antero-posterior group which passes into the grey matter.

Gehuchten's researches upon the posterior roots support the statement that some of these root fibres have their origin in cells in the grey matter of the cord. This, we have seen, is opposed to the views of Golgi, Ramón y Cajal, and Kölliker. The great

majority arise from cells in the posterior ganglia, and divide into ascending and descending branches. Both give off collaterals, which pass into the grey matter. A great many of these branches end in close relation to the cells of Clarke's column. Many of the fibres themselves end free in the grey matter at all levels of the cord, but others terminate in the nuclei of Goll and Burdach. The longitudinal fibres of the antero-lateral column behave just as those of the posterior—*i.e.*, that at variable distances they give off collateral branches, which pass into the grey matter and end free. All the nerve fibrillæ in the grey matter form a complex network. They remain quite independent of each other. Collaterals of the lateral column fibres have been seen to cross by the posterior commissure, and end in the grey matter of the opposite posterior horn.

The study of the structure of the spinal cord confirms what Golgi originally noted—that the cells do *not* anastomose through their protoplasmic processes. Each cell with its processes forms an independent autonomous structure. His has shown from his embryological studies that every nerve cell, with its processes, is formed by the transformation of a neuroblast. Nerve cells vary immensely in size, form, and richness of processes; but there is one constant structure, the axis-cylinder prolongation. It arises directly from the body of a cell, or from one of the expansions. It is distinguished by the cleanness of its contours and its regular course. It retains an unaltered diameter for a long distance; it bifurcates and gives off collaterals. It terminates blindly in one, two, or more branchlets. One can therefore divide these elements into two main sets—one occupying the top of the cerebro-spinal axis, whose process conducts centrifugally; and the other the lower part, whose process conducts centripetally. But an intermediate class exists, whose axis-cylinder process bifurcates and conducts upwards and downwards ("mixed cells"). An important fact in nerve physiology is therefore confirmed—that the axis-cylinder process of a nerve cell is a conductor, conveying any form of impression, and transmitting it, by collaterals or terminals, to corresponding structures with which it comes in contact.

Dr. Luigi Sala deals with the *minute anatomy of the pes hippocampi* elucidated by Golgi's method. The method shows that the structure of this portion of the brain is not so complicated as one would imagine from the descriptions of Kupffer, Meynert, Huguenin, Krause, and others.

Golgi divides the pes hippocampi into the four following layers:—

1. The inner or first layer of nerve fibres (alveus).
2. The grey convoluted layer, or layer of large ganglion cells.
3. The outer or second layer of nerve fibres (lamina nuclearis).
4. The layer of small ganglion cells (fascia dentata). Sala's description is based upon these four layers.

1. *The Alveus.*—This consists of a layer of thin medullated nerve-fibres running parallel to each other, and forming a bundle which covers the whole cornu ammonis. The fibres arise from the large cells of the second layer, from the small cells of the fascia dentata, and from the cells of the gyrus hippocampi. Numerous glia cells are scattered among the fibres, and here, as elsewhere, they are attached by their processes to the walls of the blood-vessels. In addition, a few nerve-cells have been found, whose axis-cylinder process finds its way into the subjacent grey layer. Golgi believed that these cells partly belonged to the grey layer. The alveus is in relation on its free surface with the cells of the ependyma ventriculorum. The bodies of these cells are continued into the alveus by a long process, which, contrary to previous belief, divides shortly after its origin into numerous branches that are attached to the blood-vessel walls and to the glia cells already mentioned, while others lose themselves in the nerve substance, apparently passing as far as the large pyramidal cell layer.

2. *The Grey Convoluted Layer.*—In addition to the characteristic cells of this layer, numerous medullated nerve-fibres are present. They consist of fibres which arise from the lamina nuclearis, and are in transit through this layer on their way to the alveus and the fimbria, and partly also of fibres which pass from the fascia dentata to the alveus. Medullated fibres are also present where this layer bounds the lamina nuclearis. The cells of the layer are pyramidal, and are continued inwards by a protoplasmic process which, dividing and subdividing, ends at the numerous glia cells scattered throughout the layer, but especially in its deeper parts near the lamina nuclearis. Filaments from these processes pass through the alveus to fuse with the cells of the ependyma. The cells occupy one or more rows near the outer limit of the layer. The nerve-process passes off from the side next the alveus into which it goes: a short distance from the body it gives off filaments, which form a delicate network in the grey layer. From this there can be traced many fibrils which go on the one hand into the alveus, on the other into the next layer.

3. *Lamina Nuclearis.*—This consists of medullated nerve-fibres arising similarly to those of the alveus. Running parallel

to the grey layer, they turn backwards with this layer and are continued behind into the white matter, which covers the subiculum and gyrus hippocampi.

4. *Fascia Dentata.*—Of this there are two layers, which must be separately considered—a superficial, formed of medullated nerve-fibres, and a deep one, composed of the characteristic cells and their processes.

(*a*) The superficial white layer of the fascia dentata consists of fibres similar to those of the alveus and the lamina nuclearis. It commences in a series of medullated fibrils, running together to form a bundle, which is most evident where the fascia dentata is folded backwards; further on it is lost in the superficial white bundle of the lamina nuclearis. Although closely associated, these two layers can be separated by the presence of numerous blood-vessels which lie between them. The fibres arise mainly from the small spherical cells of the region, and to a less degree from the large pyramidal cells.

In the part of the fascia dentata which remains uncovered, one readily sees that the superficial white layer is not at the periphery of the fascia itself, but that between this and the bundle there is a thin layer which contains no nerve-fibres. This is at the place where the fascia is in relation with the subiculum cornu ammonis. Golgi's method shows that this area is very rich in neuroglia cells, in which many of the protoplasmic processes of the small spherical cells end. It contains also a few nerve-cells, whose nerve-processes are lost in numerous fine branches.

(*b*) The characteristic cells of the fascia dentata are small and spherical, or oval in shape. They lie in the deeper parts of the layer, where they usually form a single row. They present a great likeness to Purkinje's cells—*i.e.*, from one side there spring the protoplasmic processes, from the other the single nerve-process. The former, five or six in number, branch dichotomously, and passing through the white layer, reach the neuroglia cells already described. The nerve-fibre process gives off lateral branches, which in their turn divide, and so come to form a delicate network which exists throughout the whole course of the fascia dentata. From this network numerous secondary processes arise, which are continued as nerve-fibres (type 2), but the axis-cylinder process can also be traced in many cases through the network without losing its individuality (type 1). From this network these fibres pass away to take part in the formation of the alveus and the fimbria, and to a less degree in the lamina nuclearis and superficial white layer. As long as the fibres are in

the network they remain non-medullated, and possess peculiar triangular and oval swellings.

In the network a considerable number of neuroglia cells are found, as well as isolated nerve-cells. These latter are present mainly in the outer peripheral part of the net, and possess protoplasmic and nerve processes. It is probable that these cells belong to the terminal portion of the grey convoluted layer.

1. *Some General Facts brought out by Golgi's Method.*—Of the many processes which a nerve-cell gives off, only one—the axis-cylinder process—is in direct relation with a nerve-fibre; the others—protoplasmic processes—mainly subserve the nutrition of the nerve elements through a direct relationship with neuroglia cells and blood-vessels.

2. The axis-cylinder process is related to nerve-fibres in one of two ways:—(*a*) In the first, or motor type, this process passes directly into a nerve-fibre in spite of lateral ramification. (*b*) In the second or sensory type, a network of nerve-fibrils intervenes between the cell and the nerve-fibre.

3. The protoplasmic processes are neither continuous with nerve-fibres nor anastomose with them. They probably functionate by contact with other such processes, and so conduct from cell to cell. The following facts support the belief that the protoplasmic processes are essentially of a "nervous" nature (Kölliker). (*a*) In structure and chemical constitution they are similar to the axis-cylinder process of nerve-cells. (*b*) It is not uncommon for a nerve-process to arise from protoplasmic processes.

4. *Continuity of structure is not essential for the propagation of nerve impulses; cells and fibres may functionate by contact only.*—This is probably the most important fact yet pointed out from the study of the nerve elements by this method. Kölliker has shown that this occurs in the following situations:—(*a*) In the sensory root fibres, which end free in the grey matter of the cord and medulla. (*b*) At the terminations of the lateral branches of the nerve process of many of the cells of the grey matter. (*c*) At the terminations of the longitudinal fibres and collaterals of the anterior and lateral pyramidal tracts in the grey matter of the anterior horns.

It is, therefore, apparent from these observations that the three most important functions of the spinal cord, the propagation of motor, sensory, and reflex impressions, takes place not by a direct continuation of nerve structures, but by mere contact of nerve-cells, nerve-fibres, and their processes.

WILLIAM ALDREN TURNER.

BIBLIOGRAPHY.

The following list contains the PRINCIPAL *writings of this subject.*

GOLGI. "Studi istologici sul midollo spinale," Milano, 1881.

GOLGI. "Sulla fina anatomia degli Organi Centrali del syst. nervos." 1886.

RAMÓN Y CAJAL (of Barcelona). "Rivista trimestrial de Histología," Marzo, 1889.

RAMÓN Y CAJAL. "La medicina practica," 1889.

RAMÓN Y CAJAL. "Nuevas observ. e. l. estructura de la Médulla espinal de los Mamiferos," 1890.

RAMÓN Y CAJAL. "Sur la structure de l'écorce cérébrale de quelques mammifères," *La cellule*, tome vii., fasc. 1, p. 125.

RAMÓN Y CAJAL. *Anat. Anzeiger*, 1890, Nos. 21, 22.

RAMÓN Y CAJAL. "Nuevo concepto de la Histologia de los centros Nerviosos," Barcelona, 1893.

LENHOSSEK. *Fortschritte der Medecino*, August, 1892, *et seq.*, reproduced in pamphlet form.

KÖLLIKER. "Zeitschrift fur wissensch. Zoologie," Bd. 49, Hft. 4 and Bd. 51, Hft. 1, 1890.

KÖLLIKER. *Verhandlungen der Anatomischen Gesellschaft*, Mai, 1881.

KÖLLIKER. *Anat. Anzeiger*, 1891, Nos. 14, 15.

LUIGI SALA. "Zeitschrift f. wissensch. Zoologie," Bd. 52, Hft. 1, S. 18.

VAN GEHUCHTEN. *La cellule*, tome vii., fasc. 1, p. 125.

Reviews and Notices of Books.

A Dictionary of Psychological Medicine, giving the definition, etymology and synonyms of the terms used in medical psychology with the symptoms, treatment, and pathology of insanity and the law of lunacy in Great Britain and Ireland, edited by Dr. HACK TUKE, M.D., LL.D. 2 vols., 8vo. 1477 pp. London, Churchill, 1892.

Dr. Hack Tuke has just brought to a successful issue a great enterprise which does the highest credit to English medical and psychological science. He has brought together in the form of a dictionary all the facts and ideas necessary to the scientific study, as well as to the practice, of mental medicine. Such a work could not come in better time, for at present these subjects, formerly somewhat neglected or despised in literature and science, are attracting the attention of all. It is no longer possible to deal with philosophical, medical, legal, and even literary subjects without paying regard to the researches of scientific psychologists of all countries. It will be a great help to find in the convenient form of a dictionary numerous references otherwise difficult to get at.

No one was better qualified for the undertaking than Dr. Tuke; his well-known writings on psychiatrics, his work on "Mind and Body," which already contained the germ of more recent researches gave him the necessary authority to lead the numerous and indispensable collaborators. I may be allowed to add that his simple and cordial *amabilité*, the affectionate feelings which he naturally inspires to all those who have known him, have allowed to bring together around him a large number of writers very different from one another.

These collaborators are indeed numerous: 128, and of all countries. Of them 84 are British, 17 French, 12 German, the others American, Dutch, Swiss, Italian, Austrian, Belgian, and Russian. Hence we may say that the dictionary is not a purely English work, but that it is a resultant of thought in different counties. Among the chief authors, we must mention Dr.

Urquhart, who presided over the bibliographical part, Drs. Pietersen and Willoughby, who drew up the short definitions of terms and the etymologies, met with in such large numbers. These data form one of the most useful parts of the book, and give a multitude of precise indications to the searcher. Perhaps I may here express a regret: Would it have been impossible to indicate by the side of every new and rare term, the name of the author who first propounded it, and the place where he did so?

The information given in the body of the work is completed by tables placed at the end of the second volume, by lists of the leading psychological journals and societies in different countries, by an index of legal abbreviations, etc. In a word, one is able to find immediately the practical data, without which it is at the present time so difficult to treat a scientific question.

The dictionary is moreover a complete treatise of psychiatrics, and the chief articles it contains may be grouped under five heads: 1, Historical; 2, Pathological and descriptive; 3, Anatomical and Physiological; 4, Psychological; 5, Legislative.

1. *History of Mental Medicine.*—The scientific study of lunatics appears to have a recent origin; we are too much inclined to believe that it began in the last century. It is not superfluous to remark therefore that in early times the examination of such cases had been undertaken and that some old authors have come near our most recent theories. Dr. Tuke presents these historical researches in an important article which prefaces his first volume. We are made to witness the evolution of lunacy through the centuries; there always was madness among men, because the organ of thought has always been exposed to numerous perturbing influences. There are scanty, but clear, documents concerning insanity among the Egyptians, the Persians, the Hebrews, the Greeks and the Romans. The madness of Hercules, as given by Appollodorus, is an accurate description of a typical case of epileptic delirium. Hippocrates, in the fifth century B.C., spoke of lunatics in a remarkable manner, and knew well how to connect their symptoms to cerebral disturbances. Asclepiades, Themiston, Celsus do not understand their cases so well; the latter advises brutal methods of treatment, too often followed since. Cœlius Aurelianus denounces these useless and culpable violences. "Here was a physician far in advance of all who had preceded him, and the marvel is that he should have been equally in advance of almost all who succeeded him. We desire to do him all honour and to render him the homage which is so justly his due by placing him on the

highest pedestal in the temple of fame amongst the physicians of antiquity so far as related to the medical and moral treatment of the insane."

Let us mention, in the middle ages, Paracelsus, Felix Plater, Sennert; in the 18th century, Prochaska Sydenham, and Willis, a clergyman, who undertook the cure of King George III.; we then read the epoch which Dr. Tuke hails as a new era, when Pinel at Bicêtre, and William Tuke at York Retreat, brought about a thorough revolution in the treatment of the insane.

Dr. Tuke breaks off his history of insanity at that period; but this history is supplemented by a series of papers on the organisation of asylums, and on the legislation of the insane in the principal countries.

The inquiries concerning Germany, America, Great Britain, Scotland, Spain, France, the colony of Gheel, Holland, Italy, Russia, Scandinavia, Switzerland, Turkey, brought to light many valuable documents. But it appears to us that these articles rather lose sight of medical studies.

We would have been interested in following the history of these studies in England after the time of William Tuke, and in France after those of Pinel and Esquirol; Moreau Morel, and Baillarger, a generation more engrossed, perhaps, by psychological questions, than our contemporaries. It would also have been interesting to briefly recapitulate the history of the spiritualistic school of Germany, of Stahl, Heimroth, Ideler, etc. These expressions of regret are prompted by the lively interest which Dr. Tuke's historical sketches have had for us, and by our wish to see him expanding this history of insanity.

2. *Mental Pathology.*—The first, and perhaps the most intricate, of the questions which we meet in our investigations of insanity is that of the classification of the various mental maladies. None of these classifications is perfect, and it is probable that none of them will be long-lived, but it is at least useful to know the chief ones.

Dr. Tuke, in a paper dealing with the subject, points out the classifications which are best known, or at least, most usual, in England. It would perhaps have been as well to indicate, and to more fully discuss, some rather curious classifications adopted by modern German and French authors.

Another paper deals with the diagnosis of mental diseases, and the psychological examination to which a patient should be subjected; this examination is rather vaguely defined, perhaps for the reason that even the normal psychological functions are not defined with sufficient precision.

Some of the descriptions of mental diseases have been elaborated with great care. The definition of these, often a difficult task, is summed up with great precision, the symptoms are described in an abridged form, but nevertheless in a manner sufficient for the purpose; the diagnosis, the prognosis, the pathological anatomy, and the treatment of each disease occupy a special paragraph; for instance, melancholia, and mania under all forms, are described in several articles.

We do not entirely agree with the author as regards the prognosis he gives of hysterical mania; it is customary, in medical language, to assign a very favourable sense to the term "hysterical," and to make any mental aberration that may be called "hysterical," appear as of a benign nature. This is far from being always correct; the hysteria which tends to assume the form of a characterised psychosis, is, in most cases, very serious. The first attack of hysterical mania may possibly pass away, leaving behind nothing worse than great mental debility. But it usually reappears ere long and according to Prof. Falret, whose opinion based upon ample observation, hysterical mania is one of the chief causes from which precocious dementia arises.

Dementia, which occupies so important a place in the history of mental diseases, is analysed with full detail by Dr. T. C. Shaw; paralytic dementia or general progressive paralysis is discussed by Dr. T. Mickle, who not only sums up all our present knowledge, but carefully points out all problems to be still elucidated.

Dr. Tuke endeavours to explain the somewhat obscure meaning of the word, "paranoia," and Dr. V. Parant presents a general outline of the delirium of persecution. We now reach those interesting forms of insanity where the mind does not seem to be entirely diseased, where the patient retains at least a partial consciousness of his condition, and where only a restricted portion of his thoughts seems to be affected; it is somewhat difficult to consider such maladies as insanity, properly speaking, and one is inclined to dissimulate them under such terms as neurosis, emotional neurosis, neurasthenia, or hysteria. We should not be misled by these appellations, for we have to deal with real diseases of the whole mind when we meet these forms of insanity. The various monomanias which play so conspicuous a part in the psychoses, are all separately described according to their importance.

There are interesting articles on pyromania, dipsomania, morphiomana, suicidal mania, homicidal monomania, which is

well defined in the following terms: "It is a craving for the commission of homicide, without any intellectual or passional derangement and with persistent consciousness, and anxious struggle against the besetting impulse, and, finally, after committing the deed, a lasting and consecutive moral depression." Contagious or communicated insanity, moral insanity, the insanity of denial, the delirium of doubt, may likewise be annexed to this group. The greater part of these forms, which are handled by Dr. Legrain and other authors, are presented as symptoms forming part of one great morbid type, that of hereditary insanity, and bear the title of Stigmata of mental degenerates.

As a matter of fact this theory has been for some years past expounded in various countries, and principally in France, by Prof. Magnen and his school; but, nowadays, this way of representing matters has been pretty sharply criticised. I regret that in the dictionary there are not found sufficient evidences of those discussions, which ought to have been taken into consideration.

There is evidently something very rational in this theory. "These patients," says Dr. Legrain, "retain consciousness, but they show a fundamental derangement of the mind by which the impulse is accounted for." Doubtless, the fixed idea, like the suggestion to which it is near akin, is not a primitive phenomenon. I mean that it cannot be the *first* pathological phenomenon, and that the fixed idea cannot grow up in a mind wholly sound in all other respects. The fixed idea is a kind of parasite which develops itself in a complete and isolated manner, apart from the mass of other ideas, and, as it were, independently of personal consciousness. In order to render a similar development possible, it is necessary that there should be a special state of mind allowing such an isolation, such a separation of thought; and the theory of degeneracy seems to us to be incontrovertible on this point.

But let us now examine the nature of the mental perversion which admits of suggestion and a fixed idea. Will it be sufficient to simply assert that it *is* a degeneracy, and that it depends upon heredity, which, like a *deus ex machina*, steps in and explains everything? I believe that this expression, if not absolutely incorrect, is at least far from being precise. In the first instance, heredity intervenes in nearly all mental disorders, almost without exception; it produces idiots, epileptics and the victims of typical persecutional hallucinations.

It must be admitted that the latter have insanity among their ancestors, and that they display the stigmata of degeneracy just

as much as the victims of obsessions and doubts. Why should this influence of hereditary degeneracy be confined to a special category of patients? On the other hand, this weakening of mind which makes fixed ideas possible may be due to many other causes besides heredity properly so called, *e.g.*, contagious maladies, such as typhoid fever, organic debility, starvation, overwork, emotional strain. The patients fail to properly concentrate or synthetise their thoughts; they become susceptible of suggestions and predisposed to fixed ideas for a period of more or less duration. This is especially the case with the "neurotic" state. It is daily becoming more and more recognised that neuroses, neurasthenia, and hysteria, are the real soil upon which doubts, interrogations, obsessions and impulses, take root and grow. Doubt exists with the majority of such patients, generally bearing upon all sensations, and upon all images which are badly perceived and synthetised in the mind. This doubt may easily become fixed upon some particular point, it may assume the shape of obsessive interrogations, and develop into a downright delirium.

Are we then to designate all these mental disorders as degeneracies? Yes, possibly, in one sense; for it is evident that a neurasthenic patient has not the mental power which he ought to have, and that he is in a fallen degenerate state. But then the word loses all its precision, for it thus becomes applicable to almost all maladies without exception.

I do not intend to discuss here this problem of classification, but simply wish to point out its difficulties. It matters little, in reality, what designation we use; and it is sufficient to establish the fact, as has been done by Dr. Legrain, that fixed ideas originate in a predisposition of the mind, and to carefully analyse that mental condition.

Dr. Tuke is so thoroughly convinced of this necessity, that he does not hesitate to devote several exhaustive paragraphs to the study of neuroses. Not long ago, hysteria was described along with the organic disorders of the cord, disseminated sclerosis, and tabes, but it is far more correct to classify it among the psychoses. I even believe that it is destined to play a very important part in works on mental medicine; for it is simply an insanity the phenomena of which are accessible to direct observation and analysis and even to a process of experimentation.

We must therefore accurately ascertain the mental condition of hysterical patients, before entering upon the study of more complicated forms of insanity.

The description of hysteria is given from a clinical standpoint

in an elaborate article by Profs. Charcot and Marie. I do not think that it would be right to look upon it as giving the French theory on hysteria. It is not, in fact, a theory; but rather a description of the attacks, of anæsthesiæ of contractures, such as are met with everywhere, even in England. Nevertheless, we have been greatly interested in another paper on hysteria by Dr. H. B. Donkin, which, we are told, represents the opinion of the medical faculty in England regarding this neurosis. The clinical description differs very little from the preceding one; Dr. Donkin lays less stress upon the attack, the serious nature of which is rather lightly touched upon, but, greatly to our satisfaction, he insists rather more upon the psychological interpretation of hysteria. "There is much less risk of error in roughly classing hysteria as a species of insanity than in taking one of its physical expressions, the hysterical fit, for instance, or sexual disorder, as its central fact. . . . One French school of neurologists, who have worked so exhaustively and minutely in this field, have of late espoused the long prevalent English view of the *rationale* of these phenomena, some of its most modern opponents ably illustrating many hysterical phenomena by the facts of hypnotism, and indeed claiming *suggestibilité* as the essence of the hysterical neurosis."

I should have been glad to know the English authors who have clearly demonstrated the psychical nature of hysterical accidents, prior to the book of Briquet, who considers hysteria as an emotional malady, and to the teachings of Charcot concerning traumatic paralysis; but I am happy to learn that this opinion has so long been held in England.

Further on Dr. Donkin has, it seems to us, somewhat lost sight of the method which he had so correctly indicated at the beginning. No doubt he distinctly recalls the theory of psychical paralysis "dependent on idea." He also points out the lack of effort, the minimised will-power of the patient who does not even endeavour to move, but he scarcely gives us a psychological explanation of the other phenomena, of the anæsthesias, contractures, convulsive attacks, etc. The disturbances of memory, of volition, of attention, and of emotion might be indicated with greater precision; but these slight wants are of minor importance, and we do not dwell upon them, since, on the whole, the conception of hysteria seems to us to be very accurate.

Our thanks are due to Dr. Donkin for the clearness with which he expresses himself concerning somnambulists and the mediums: "It is among hysterical sufferers that many instances

of natural somnambulism and most of the subjects of induced hypnotism are found. Notwithstanding the recent teaching that all human beings are potentially hypnotisable, it is certain, from general experience, that human beings are hypnotisable in direct proportion to their nervous instability." These are words which it would be well to repeat in England, where so many authors still represent somnambulism and automatic writing as being a normal characteristic of people of sound mind.

We pass over numerous articles on the mental and nervous pathology to call attention to a paper on neurasthenia, and a series of fine essays on the real degenerates, imbeciles, idiots, etc., considered from a physical and moral standpoint.

The reading of all these articles gives, if not a complete knowledge of, at least a clear insight into, mental pathology and the controversies raised by the investigation of the subject.

3. *Anatomy, Physiology, General Medicine.*—Although we are yet far from understanding fully mental diseases, we are at least able, nowadays, to study them by different methods. Anatomy, physiology and general medicine, each furnish facts and theories which enable us to arrive at an improved interpretation of certain forms of insanity. We note in the dictionary the articles by Prof. Benedikt on cerebral craniometry, the description of cranial modifications observed in micro-cephalic cases, the very useful information about anatomical preparations and microscopical examination of nervous tissues. The anatomy of the brain, of its membranes and its vessels, is described with great precision by Dr. A. Bruce, and forms the subject of a new supplementary paragraph at the end of the second volume.

A question of importance in our days, the morphology of cerebral convolutions, is treated in an elaborate article by Dr. Hack Tuke; several accurate figures accompany the description. We also find information relating to the specific weight of the brain, to its chemical constitution, and to its various modifications in insanity.

Passing from anatomy to physiology, we are met by the first and most important of all the problems, that is, the formation of the new being whether in health or disease; this formation depends upon the healthy or sickly disposition of the parents, upon heredity in short; the study of Heredity, by Dr. C. Mercier, is embodied in one of the finest articles of the dictionary. Its nature is not discussed (the theories of Weissmann are scarcely touched upon), but it is investigated from a practical point of view in its relations to mental aberration. Some of the most

incontrovertible laws of heredity, such as the influence of consanguinity, the influence of the mutual suitability of the parents (an influence as important as it is little known) progressive heredity, the main feature of which consists in an increased accentuation of the malady in each successive generation, and also that species of heredity which may be called regressive, and operates in an inverse ratio, the manifestations of the disease gradually decreasing and showing a tendency to the re-establishment of the normal type,—all these problems are there discussed in a most interesting manner and can only be alluded to here.

If we follow the development of a child generated under such conditions, we find that it passes through different stages, each of which is beset by danger. Dr. T. S. Clouston, in a very original essay, gives us an insight into "developmental insanities and psychoses, the delirium and night terrors of children, the insanity of puberty and adolescence." Between the ages of 15 to 25 years the development of man is complete, and it is during this decade that hereditary influences assert themselves in the most dangerous manner, and that most of the neuroses begin to show themselves. All other physiological data are reviewed in a like manner, *e.g.*, the height, the conformation of the head, the weight, and even the peculiar character of the hair are successively examined in order to investigate the modifications produced in the cases of the insane. The physiognomy of the sufferers forms the theme of an interesting description, which is rendered more accurate by photographic reproductions. Surprising asymmetries of the face will be discovered, caused by the separate contraction of one of the lateral parts of the fronto-occipital muscle; the modifications of circulation are carefully examined (pulse in insanity, use of the sphygmograph in the various forms of insanity). The temperature of the body, that of the head, the composition of the blood, the saliva, and the well-known phenomenon of excessive salivation, the urine, the bladder, and the singular influence which thought exercises upon the latter, the sexual functions, the periods and the periodical phenomena which appear to be connected with them, the troubles of puerperality of menopause—all these phenomena play an important part in mental pathology, and are reviewed in succession. The functions of the nervous system naturally occupy a large space, the reflexes, the pupillary reactions, and the examination of the eye in the insane, sleep, sleeplessness, are essays which we may here refer to more particularly. They doubtlessly offer many debatable points, but

they clearly bring out the difficulties to be surmounted, and the importance of the problems. Finally, the physiology of the brain, from our present standpoint, occupies a prominent position. A very exhaustive article by Dr. Beevor, describes the functions of the cortex, internal capsule, corona radiator, ganglia, cerebellum, corpora quadrigemnia. The experiments of Hitzig, of Ferrier, the clinical studies of Charcot and of Hughlings Jackson, are mentioned, but the author has apparently undervalued the works of the anatomo-pathological school, who, on this point, as well as on many others, have largely added to the work of the physiologists. We cannot examine the great share which, in this dictionary, has been allotted to general medicine; the very numerous diseases which may influence insanity are carefully studied. We will only recall the fine articles on alcoholism and the various forms of intoxication; on the various infections, malaria, influenza, syphilis, tuberculosis, and typhoid fever, the importance of which, from a mental point of view, could, in my opinion, have been somewhat more clearly indicated.

Not only are neuroses considered as causes of insanity, but some of the authors, I think quite correctly look upon them as insanity itself. All these different essays tend towards the same point, viz.: the analysis of mental disturbances and of their physiological conditions.

4. *Psychology.*—Mental diseases are more complex than others and cannot be studied in the same simple manner as maladies of the body; they present two different aspects, the one is external or physical, the other is internal or psychological; it is not sufficient to describe an insane patient externally, and to demonstrate the modifications of the pulse or the urine, we must penetrate the depths of his mind and must picture to ourselves, as much as possible what he feels and what he thinks; psychological research is absolutely indispensable to the specialist in insanity; the specialists in lunacy of the time of Esquirol, of Moreau (de Tours), and of Griesinger were quite conscious of this fact; but for some time the writers who succeeded seemed to neglect those studies, and we are glad to note that these researches are now being resumed in every quarter. Is not the number and the intrinsic merit of the paragraphs devoted to pure psychology in a dictionary of mental medicine, a sign of this interesting movement, of this return to mental studies by physicians for the insane? At the beginning of the work, as a preface which was considered necessary, there will be found a long article by Mr. Coupland upon the "Philosophy of Mind"; if, on the one hand, we regret to find that this work

has not a more "medical" tendency, and that it does not lay greater stress upon pathology, on the other, we are glad to see that that the profession in England attach great importance to purely theoretical psychology. In the body of the dictionary itself, we meet with numerous articles upon the same subject, "Psychological Methods," by Dr. H. Donaldson, A. D. Waller, W. Bevan Lewis; "Psychosis," by Dr. Francis Warner; "Consciousness," by Dr. C. Mercier; "Reaction-time," by Prof. Joseph Jastrow; "Memory," by Drs. Bleuler, Ribot and Willoughby; "Attention," by Prof. James Sully; "Dreams and Hallucinations," by Dr. Hack Tuke; "Instinct," by Prof. Romanes; "Actions and Behaviour of the Insane," by Dr. C. Mercier; "Hypnotism," by Drs. Charcot, Gilles de la Tourette, Myers, etc., etc.

To review in detail all these articles would be beyond our limits, we can only venture upon a few remarks regarding some of them in order to show how much importance we ascribe to them. Let us first of all point out the principal attempts at psychology which are anterior to our epoch. Mr. Coupland justly thinks that the history of a science is conducive to the knowledge of the science itself, and he gives us some interesting information concerning Aristotle, Locke, Hartley, James Mill, etc. I hope I may be excused for passing a slight criticism, or rather for putting a simple question. Does the author seriously believe that psychology has been studied in Greece and in England only? Does he think that psychological study has descended direct from Aristotle to Locke and Hartley? Have such men as Descartes, Malebranche or Leibnitz never existed? Here we have, I think, a very serious historical question, for psychological physiology, as understood nowadays, proceed far more directly from the Cartesians than from Locke. The influence of the French philosophers of the seventeenth century was considerable, and it can easily be shown that the English psychologists, as, for instance, Dr. Hume, of whom Mr. Coupland does not make sufficient mention, have been inspired by the works of that school. Nor do I believe that German psychology begins with Herbart, whom Mr. Coupland very tardily mentions after the whole development of English psychology; it is considerably anterior and dates back as far as Leibnitz, whose theories, even in our days, play so important a part in psychological pathology.

Mr. Coupland is, no doubt, of the same opinion as myself, but perhaps he intended to present only a brief summary, and will, at any rate, excuse my criticism.

It is interesting to observe that all authors who nowadays

devote themselves to physiological psychology are nearly agreed upon one general idea. The old theories upon the nature of psychical facts, upon their connection with the organism, the old metaphysical theories concerning spiritualism or materialism, properly so-called, are no longer held. The doctrine of correspondence, of concomitance between psychological and physiological phenomena, as Dr. H. Jackson observes, is sufficient for the present purposes of science. "There are," as Mr. Coupland pithily remarks, "either two sets of phenomena, heterogenous but concomitant, or one phenomenon describable in two forms of language according as it is regarded as strictly limited to one individual, or as capable of becoming a portion of universal experience." This conception seems to us to be correct; sufficient, at least, to allow experimental researches, without unduly embarassing us by metaphysical problems. But it should be properly understood, and we should accurately guage the depths into which it may lead us; if the physical and mental phenomena form two series of parallel facts, it will be necessary to study each series separately with the process adapted to it; we must study the former by physiological methods, and must endeavour to reach the psychological facts by introspection, by observation of actions and language, by time measurement, &c. It is only after a complete and independent investigation that these two series of facts should be brought into juxtaposition. A physiological fact which has been clearly demonstrated by suitable methods, may then be correctly and usefully collated with a psychological fact which has been equally well proved by the proper process. If there is an error which seems intolerable to us, it is to invent, to construct from imagination either of these two facts and to bring it into juxtaposition with the other. Some authors delight in explaining a moral fact by imagining some physiological process or other, which they then collate with a psychical phenomenon. Thus, in the case of an association of ideas, or other very definite psychological fact, instead of accurately describing its exact nature they speak of an intercommunication between nervous fibres, which takes place at that moment, of contact between branches of nerve cells, of a nervous current which has been established, etc., etc. Has this communication, this contact, this current, been observed by them through the microscope at the time of its occurrence, or have they even ascertained the existence of an association of ideas? If not, then why do they mention it? Such modes of expressing oneself are mere, meaningless, verbal translations. The actual psychological fact is made to disappear by translating

it into another language; nothing is added to our real knowledge, and it would be as well to speak in poetical metaphors as to indulge in this vainest and hollowest of metaphysics.

We may now enter upon the detailed study of psychological methods, most of which are well analysed in the dictionary. Attention may be called to the work of Dr. Fr. Warner under the title of "Psychosis," and to the methods he employs in order to register movements and to interpret them from a psychological point of view. Several authors, particularly Mr. Coupland, insist upon the necessity of studying social life in order to arrive at a correct interpretation of mental facts. "An individualistic psychology, aided by all the resources of the psychical laboratory or clinical experience, would be but a maimed and incomplete psychology; mind cannot be comprehended without a study of its development, and the development of mind implies the action and interaction of minds." Each of these various methods must complete the other.

Consciousness has been studied from various standpoints, for instance, it has been analysed in itself, in its psychological nature, and in its connection with physiological phenomena. It seems to us that in the first essay the author greatly complicates the problem; he speaks of all the phenomena of thought, of comparisons, of ratiocination, of scientific knowledge, which is not a fact of simple consciousness, but is superadded to consciousness properly so called. On the other hand, it appears that the author does not sufficiently take into account the different varieties of consciousness itself. The elementary consciousness, which attends the simplest psychological facts, and the personal consciousness where the idea of the "ego" plays the principal part, should not be absolutely confounded. This distinction is particularly useful for the interpretation of pathological phenomena.

In the second essay the physiological conditions of consciousness are carefully analysed; it is shown that consciousness does not exist without nervous action, but that the reverse is not true and that nervous action may, in appearance at least, sometimes be produced without consciousness. The distinction between the different degrees of consciousness could fairly be applied to this discussion. Authors perhaps too easily adopt the theory of "epiphenomenal consciousness," and do not sufficiently observe that this theory is at variance with their fundamental principle concerning the correspondence which exists between psychological and physiological facts. We will not insist upon the

description of the disturbances of consciousness; it is given in the dictionary in a very elementary manner. Let us merely express our amazement at finding that the facts of the double of consciousness, and even the obsessions of lucid madness are being unhesitatingly connected with cerebral duality. For more than ten years these somewhat childish interpretations have been attacked in France and in England, why then are all objections passed over in silence? Let us also express our regret at finding that the numerous researches on subconscious phenomena, and the overwhelming part they play in mental pathology are barely hinted at. Possibly the authors did not deem it expedient to enter deeply into the studies on the pathological alterations of consciousness, and so confined themselves to the description of well-known facts without entering into analyses which seemed to them, perhaps with some reason, still resting upon too hypothetical a basis to occupy a place in a dictionary. The sensations are repeatedly analysed, their laws, their conditions, and their principal modifications are graphically described; the combinations which go to form cœnesthesis, the secondary sensations which produce the well-known phenomenon of "coloured audition," form the subject of interesting articles. The discussions might be more complete, but the leading ideas are sufficiently indicated. But why did the authors shrink from investigating the problem of muscular, or kinesthetic, feeling? The laws of the association of images have been carefully studied, as was to be expected in an English work. Mr. Coupland points out, with great truth, the close analogy which exists between two laws which formerly were kept too much apart; that is, the laws of association by contiguity and by similarity. He recognizes and very clearly demonstrates that association from similarity is in reality nothing but an association from partial contiguity. Some excellent remarks on the association of ideas which formerly had not been united, and on the difficulties of those new combinations should have led him to an investigation of the phenomena of mental synthesis which, as a rule, are so little studied in England. Among the various articles on "Memory," we will call the attention of readers chiefly to a summary of the labours of Mr. Galton on visual memory; and to the processes most frequently used by calculating prodigies. The disorders of memory are well described, and we only regret that the authors did not enter more fully into their psychological interpretation.

It is well known that English psychologists never were addicted

to the study of the higher operations of the mind, such as, for instance, judgment, belief, and generalisation. They are always inclined to confound them with simpler phenomena, such as the association of ideas. This tendency appears also in the dictionary, although some exceptions must be made, *e.g.*, for Mr. Coupland's article on "Classification" (38), and especially for Prof. James Sully's remarkable essay on "Attention." Most of the recent experiments concerning attention, its duration and its intermittances, are fully summed up. The theory of M. Sully, which may be summarised thus: "Attention reduces the confused multiplicity of excitations to a relative simplicity," confirms and explains most of the known facts. It was difficult to exhaustively study "Language" in the dictionary; and only the leading ideas concerning its formation, and the disturbances which it presents in the various forms of æphasia, are indicated. But why pass so lightly over the "inner speech" and psycho-motor and Rivesthetic verbal hallucinations, which play a prominent part in mental diseases? As regards the phenomena formerly grouped together under the appellation of "active faculties," we will point out an excellent essay by Prof. Romanes on "Instinct"; some passages by Mr. Coupland on the "Emotions"; and the interesting article by Dr. C. Mercier, on "The behaviour of the insane." In this latter work we clearly perceive how the character of human activity chiefly consists in a continuous effort to adapt oneself to the complex and varied external circumstances; and how, in the mind of the insane, this power of adaptation is subject to metamorphoses and diminutions.

The study of the Will is one of the most difficult topics; too much attention is, perhaps, still bestowed on absolute problems, it would be better to minutely analyse the psychological characters of the phenomenon itself, without paying so much attention to the more or less intelligible theories concerning liberty. This task has been attempted in several articles by Mr. Coupland and Dr. Warner. Perhaps it would have been advisable to lay greater stress upon the considerable part which the idea of personality plays in voluntary actions.

Finally, the disorders of volition are dealt with in a very interesting article by Prof. Ribot; and several essays on suggestions, obsessions and impulses, complete the analysis of voluntary actions. In a rather short article, Mr. Myers tells the history of hypnotism; he lays before us certain, hitherto unknown, details concerning the development of these researches in England. Several authors describe hypnotic phenomena without, in my

opinion, sufficiently insisting upon the psychological interpretation of these phenomena, now so well-known and widely discussed.

It is hardly possible to fully review a dictionary, and still more difficult to analyse a whole psychology disseminated through its pages; we do not pretend to pass a final judgment upon the whole work, but merely to express in a few, possibly somewhat superficial, words the general impression which the reading of those articles has produced upon us.

The psychology exposed in this book is, on the whole, that of the English school, as expounded by Prof. Bain and Mr. Spencer. We do not intend here to discuss this psychology; it has advantages as well as drawbacks which are well-known; but we wish to make a general remark about it. It is a general, a theoretical psychology which is applicable to man in his normal state; it does not deal with the diseased individual for the interpretation of whom it is not adapted. Now, the alienist principally requires a pathological psychology, analogous in its main features, to normal psychology, but also laying chief stress upon the points with which he is chiefly concerned. The theories of perception, the transformation of the image into an external object, which is the starting-point of hallucinations, belief, doubt, automatism, &c., deserve a closer study from this point of view. It is not a defect in the dictionary that we are now censuring, but rather a defect in psychology itself. Let us hope that this science will become more and more practical, and more and more capable of rendering to the physician alienist such assistance as he is entitled to expect from it.

5. *Treatment and Care of the Insane.*—Pathology and psychology form only a part of this great work; specialists in lunacy will also find in it much information and many practical hints. Numerous articles refer to the treatment of the insane; among which is a very encouraging essay by Dr. Pliny Earle, on "The Curability of the Insane." Further, the articles upon the psychology of insanity, the treatment of the insane by boarding out, the construction of the asylums themselves, the general and some special methods of treatment of the insane, as for instance, treatment by electricity, which is described with great care. It is, perhaps, to be regretted that the various forms of moral treatment which play so important a part, are so vaguely indicated. It is difficult to gain an accurate knowledge of the special legislation generally very imperfect, under which lunatics are placed. English physicians will be glad to find in the dictionary all the practical information indispensable to them.

Some of the medico-legal discussions have a more general interest, such as those which refer to the responsibility of the insane. The important article by Dr. Orange on "Criminal responsibility in relation to Insanity," sums up all past discussions about this difficult question; it treats of the very controversial problem of partial monomania, and gives the reader a general idea of the principal works upon criminal legislation in its application to the insane.

Dr. Havelock Ellis's article on "Criminal Anthropology" summarises the ideas of Lombroso, of Benedikt, and of Ferri, concerning the relations between crime and insanity.

Such are the principal articles contained in this great work. We have hardly attempted to do more than to point them out to the reader, and give a summary idea of their contents, so as to demonstrate their variety, their importance, and their usefulness to psychologists, physicians and legislators.

The few criticisms in which we have indulged only tend to prove the great interest which we have taken in reading this work.

PIERRE JANET.

Illustrations of the Mid and Hind Brain. By ALEXANDER BRUCE, M.A., M.D., F.R.C.P.EDIN. Young J. Pentland, London and Edinburgh, 1892.

THIS is an elaborate work on the nerve tracts of the mid and hind brain, and the cranial nerves arising therefrom. It consists of two portions: the first is given over to a description of the author's own observations upon this portion of the brain, the reading of which is rendered easy by a number of valuable schemata: while the second part consists of a series of highly artistic coloured plates, taken from the author's original sections.

In all cases the preparations have been made from the human fœtus; and, except where otherwise stated, from fœtuses at the time of birth. To ensure accuracy in reproduction the plates were taken from the sections, which have been stained entirely by the method of Weigert, under the camera lucida.

Throughout the text there is a digest of the recent literature upon the subject.

The work is divided into two chapters; the first dealing with the cranial nerve nuclei and their connections, the second with the tracts of the medulla, pons and crura.

In the chapter devoted to the consideration of these tracts, prominence is given to certain structures which are still the object of investigation. Reference is made firstly to the upward continuation of the antero-lateral ground bundle of the spinal cord. The anterior ground bundle of the cord is displaced backwards by the inferior olive, so as to become the posterior longitudinal fasciculus of the medulla, and in one of the plates this relation is well shown, the two structures being seen in continuity in a well-planned longitudinal section. The adjacent portions of the lateral ground bundles lie in what Flechsig has designated the inner field of the *formatio reticularis* or area between the roots of the hypoglossal nerve and the raphé; while the outermost of the lateral ground bundles forms chiefly the longitudinal fibre system of the grey reticular formation. The chief of these form the "lateral medullary tract," a strand containing the antero-lateral tract of Gowers, which ends mainly in the lateral nucleus of the medulla. The author considers that the upward continuation of this tract is more likely to be settled by a study of its degeneration than of its development—a view in which we concur.

The author's observations upon the formation of the mesial fillet accord with those of other observers who have worked with this method; its termination, however, being beyond the scope of the work, is still left as a matter of doubt. A relation of some importance between the lateral fillet, and the lateral column of the spinal cord is indicated.

The restiform body is formed of two distinct portions; the external or spinal division being composed of the direct cerebellar tract, of fibres from the post pyramidal and cuneate nuclei of the same and opposite sides, the olivary-restiform fibres, and of fibres from the lateral nucleus of the medulla. The internal or medullary portion is derived from the external nucleus of the vestibular root of the auditory nerve and of a tract from the superior olivary body.

The superior cerebellar peduncle, the inferior olivary body and nucleus dentatus of the cerebellum, the corpus trapezoideum, and the flocculus are each the subject of careful study.

But the chapter upon the connections of the cranial nerves is of greater interest to physicians. Mention is therefore made of the following points which appear to be of clinical importance.

1. *The hypoglossal nucleus.*—The author has traced by longitudinal horizontal sections a fine bundle of fibres running parallel to the raphé from the hypoglossal nucleus towards the knee of the

facial nerve; while a connection between this nucleus and the internal auditory nucleus is also indicated. He confirms the observation originally made by Lockhart Clarke that fibres pass from the hypoglossal nucleus to the issuing vago-accessory nerve roots, and he thinks it probable that this nucleus forms at least part of the motor nucleus of the vagus.

2. *The ascending root of the auditory nerve* (vestibular nerve) is shown to have an important connection with the cuneate nucleus, a relation which does not appear to have been previously pointed out. By this means the vestibular nerve forms important connections with the fillet, the restiform body and the postero-external column of the spinal cord. The author negatives Edinger's view that the ascending auditory root is a "direct sensory cerebellar tract" connecting the roots of the vagus, glosso-pharyngeal and auditory nerves with the cerebellum. It is found to be continuous with and to myelinate at the same time as the anterior root of the auditory nerve.

3. *The facial nerve.*—Without bringing forward any further proof of the several nuclear origins of the fibres composing this nerve, other than that already mentioned, the author merely states the views expressed by Mendel, Gowers and others, and gives a scheme of the probable composition of the emerging root of this nerve, and indicates its tripartite formation from the oculo-motor, facial and hypoglossal nerve nuclei.

4. *The sixth nucleus.*—An elaborate diagram is given of the connections of the sixth nerve nucleus, which are stated as follows: (*a*), with the posterior longitudinal bundle of the opposite side; (*b*), with the superior olivary body; (*c*), with the internal and external auditory nuclei and the flocculus of the same side.

5. *The ascending and descending roots of the fifth nerve* are briefly described. The termination of the fibres of the former in the cells of the grey matter internal to the gelatinous substance of Rolando is indicated, but their further relations are still "*sub judice.*" A connection between the substantia gelatinosa and the cuneate nucleus is mentioned, as well as a hitherto undescribed tract of fibres coursing parallel to the ascending root, but on the mesial side of the gelatinous substance. The origin of the fibres of the descending root is stated to be as high as the upper limit of the nucleus of the third nerve.

6. Some space is given to a detailed description of the several groups of cells which form the nucleus of the *third nerve.* The author, who has carefully studied this subject, has very properly adopted a nomenclature in accordance with the position of the

several cell-groups rather than from the name of the observer who originally described them. These cell groups are: (*a*), anterior; (*b*), postero-external; (*c*), median or central; (*d*), postero-median (the pale or Edinger-Westphal nucleus; (*e*), superior (the small-celled nucleus of Darkschewitsch).

We have no hesitation in saying that this work takes high rank among the numerous treatises upon the complicated subject with which it deals. To teachers and students of neurology the book is invaluable; but we also cordially recommend it to physicians who are interested in this subject, by reason of its practical bearing on the study of diseases of the nervous system.

W. A. Turner.

Abstracts of British and Foreign Journals.

SOME RECENT PAPERS ON NEUROPHYSIOLOGY.

(1) Einthoven. Broncho-motor Action of Vagus (*Pflüger's Arch.*, 1892.) An action of nerves upon bronchial muscle has long been an article of medical faith, more especially in the theory of spasmodic asthma, but until quite recently the physiological demonstration of such an action has been very incomplete. Manometric observations of intra-pulmonary pressure have not afforded marked enough results for us to admit bronchial spasm as a really effective phenomenon; the variations of pressure observed by Bert and Franc have been insignificant in comparison with the amount of action that must be supposed to occur in a fit of asthma. The reason of this failure will be evident when we have recognised the principle of the method by which a satisfactory demonstration has recently been given by Professor Einthoven (of Leyden).

This method is as follows: A dog paralysed by curare is submitted to artificial respiration, great care being taken to inflate at regular intervals with uniform quantities of air, a side tube from the tracheal tube leads to a manometer, by way of a special tap which is automatically opened by means of the respiration apparatus for a definite short period of each respiration cycle, thus letting a short puff of pressure into the manometer. If the intra-pulmonary air pressure rises and falls regularly, the succession of puffs produces a level series of manometer effects, but if the bronchial muscle be made to contract by excitation of the peripheral end of the vagus, the puffs occur at greater pressure and the manometer rises. A constriction of minute bronchioles which might cause little or no effect upon a *steady* intra-pulmonary volume or pressure of air, will produce a very evident effect when the rising and falling phases are, so to speak, "tapped" on this "window plan," which is similar in principle to the method of "tapping" an electrical pressure by the rheotome (p. 312).

By these means Einthoven has found: (1) That the excitation of the peripheral end of either vagus causes strong bronchoconstriction, shewn by increase of pressure in and distension of

the lungs; (2) that this action is abolished by nicotin or by atropin; (3) that broncho-constriction is caused by inhalation of CO_2. The broncho-constrictor effect of vagus excitation begins in about 1.2 to 1.3 seconds, and wears off very rapidly during excitation; the effect on the manometer may amount to 175 mm. H_2O or more.

These results, as set forth in Professor Einthoven's paper, fully substantiate this method as a valuable means of studying the action of plain muscle, comparable with the blood-pressure method, and in some respects of superior simplicity.

To many readers the bearing of these results upon the theory of asthma, will be the point of greatest interest. This is dealt with in the two concluding sections of the paper, where the author urges that bronchial spasm is capable of producing acute emphysema, that the respiratory type during an asthma paroxysm is strong, short inspiration, followed by weak long expiration (and not, as usually described, by still more violent expiration), and that *possibly* the subcutaneous injection of atropin may be found of service to cut short a paroxysm. The action of stramonium vapour was not investigated.

Einthoven's paper has given rise to a considerable amount of interest in Vienna. Beer (writing from von Basch's laboratory) published a preliminary communication (*Cbt. f. Physiologie*, 1892, p. 782) as a direct consequence of that paper, and containing several similar conclusions, but laying stress upon the mechanical effects of the circulation. The same author has more recently published a paper of upwards of 100 pages in the 1892 supplement of *du Bois-Reymond's Archiv*.

Von Basch (*Pflüger's Archiv.*, vol. 52, p. 415) and Grossmann (Ibid., p. 567) vehemently criticise Einthoven's criticism of their previous papers. The criticism is very controversial in tone and very obscure in exposition, and does not affect the main point of Einthoven's paper. Grossmann reaffirms von Basch's doctrine that engorgement of the pulmonary capillaries obstructs respiration by distending the alveoli, and quotes figures from Einthoven to show that they prove what his own figures had proved; but on referring to the explanation of them given by Einthoven, and omitted by Grossmann, it will be found that there is no real similarity either in the figures or in the conclusions drawn from them.

(2) **Heese. Influence of the Sympathetic upon the Eye, more particularly upon the Movements of the Iris** (*Pflüger's Arch.*, 1892). The vexed question whether dilatation of the pupil

by excitation of the sympathetic is effected by active contraction of a radially disposed *dilatator pupillæ* or by passive relaxation of the constrictor pupillæ, appears to be finally set at rest by the experiments of Heese. His proof may be thus shortly summed up: graphic records of radial contraction of the iris, caused by excitation of the sympathetic, after removal of the sphincter; ditto of a sector of the iris isolated by two radial incisions. Great care was taken to perfectly immobilise the eye, and the records given are typical of the contraction of smooth muscle—latent period of about 2 seconds, contraction lasting 10 to 15 seconds.

(3) **Langley and Anderson** have given expression to an identical conclusion, based upon similar data. The two papers are quite independent of each other; the date of publication of the German paper is September 16th, 1892; the dates of publication of the English papers are May 14th (communication to the Physiological Society) and October, 1892 (full paper in *Journal of Physiology*).

(4) **Nahmmacher on the Influence of Reflex and Central Excitation of the Optic Nerve upon the Position of the Cones of the Frog's Retina** (*Pflüger's Archiv.*, 1893, p. 375). The experiments on the retino-motor action of fibres in the optic nerve (of which the first results were announced by Engelmann, at the Copenhagen Congress in 1884, *Pflüger's Archiv.*, vol. xxxv.) have, on several occasions, been taken up again in the Utrecht laboratory. Grijns (as reported by Engelmann in Helmholtz's *Festschrift*, two years ago) found on the frog that the retinal current of one eye varies with actinic and chemical stimulation of the other eye. Engelmann, in the absence of any evidence of the possibility of obtaining evidence of reflex electrical variations in different nerves by excitation of other different nerves, concluded that the reflex retinal variation observed by Grijns, depends upon different fibres, *i.e.*, upon the retino-motor fibres inferred to be present in conclusion of his micro-physiological observations on the retinal cones. The observations of Gotch and Horsley to the effect that the excitation of afferent nerves may give rise to centrifugal variations along other afferent nerves, are, however, contradictory of Engelmann's negative assumption; and the experiments of Nahmmachar were undertaken to determine whether the crossed retinal effects are attributable to efferent or to afferent fibres. His conditions of experiment, and his results were briefly as follows:—Rana temporaria; July, August, and September; with and without section of an optic nerve; slight curarisation; total darkness for five

hours; chemical excitation of one retina by a crystal of common salt left in the eye for 10 m. before decapitation; eyes placed in 3½ % nitric acid. Comparison was made between retina of different frogs: (1) normal; (2) injured and not excited; (3) injured and excited; two series of experiments—the first with reflex from eye to eye, the second with direct excitation of the chiasma or optic nerve. As regards the first series, the cones were found to be retracted in thirteen out of fifteen cases of reflex excitation *versus* eight out of fifty-two control cases without excitation (viz., 86.6 *versus* 15.4 per cent.); as regards the second series, the cones were found retracted in forty-two out of fifty-one cases of direct excitation *versus* eighteen out of ninety-two control cases without excitation.

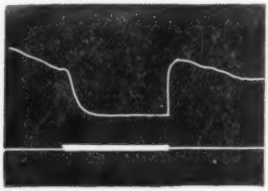
The author's conclusion is that these figures amount to a final proof that the optic nerve contains fibres, the excitation of which causes contraction of the cones of the corresponding retina, *i.e.*, presumably efferent fibres.

(5) **Fritsch,** in the account of his recent observations on electrical fishes (*Du Bois-Reymond's Archiv.*, 1892, Suppl., p. 229), makes remarks on the motor cells of the spinal cord calculated to make us pause before accepting the functional interpretations that have been lately given of the fine anatomy of nerve-cells. The electrical nerves (Mormyrus) arise as unbranched axis-cylinder processes from large multi-polar cells (100 to 500 μ) that occupy the entire transverse section of the grey matter. These unbranched processes acquire a medullary sheath while still in the cord, and are thus readily distinguished from the "protoplasmic" processes, which unite the cells into a closed plexus "as if for a common function." He proceeds to say: "This discovery gives positive proof that protoplasmic processes serve to unite nervous elements, and disproves the supposition of Golgi and of his followers, to the effect that the axis-cylinder-process alone is of 'nervous character.'"

(6) **Kaiser. Interference-phenomenon in the Nerve-Muscle Preparation** (*Zeitschrift f. Biologie*, 1892, *p.* 417, *vol.* 28). An extremely interesting experiment, exhibiting what at first sight it would appear most natural to designate as an inhibitory phenomenon, has recently been published by Kaiser. The experiment is easily repeated and appears to be a suitable one to include among practical exercises such as are drawn up for advanced students. It may therefore suitably be described in the form of an instruction to such students about to repeat it.

A nerve-muscle preparation is made, and enclosed in a moist

chamber; the muscle is connected with a recording lever and weighted by 20 grms.; the central end of the nerve is laid across ordinary platinum electrodes from an induction apparatus; a portion of the nerve nearer to the muscle is made to hang in a loop and brought into contact with strong glycerine. As the effect of this glycerine stimulus, the muscle after a time becomes tetanised; when this tetanus has fully developed an interrupted



current of moderate strength is thrown in. During its passage the tetanus is cut down, as shown in the accompanying figure taken from among the illustrations given by the author.

(7) **Piotrowski** (*Centralblatt f. Physiologie*, 1892, p. 604, *Journal of Physiology*, 1893, p. 163), working in the Cambridge Laboratory, repeats and extends Biedermann's observations on the muscles of the crayfish claw. With weak currents the abductor muscle give contraction, with strong currents relaxation. The adductor muscle does the reverse; *i.e.*, relaxes with weak and contracts with strong currents. These antitonic effects, best brought out when the disposition to tonic contraction is most pronounced, *i.e.*, in the summer, they are best demonstrated on the abductor; and in all such experiments the antagonist muscle is of course divided. By weak single shocks he obtains double effects of a similar character, viz., a preliminary relaxation preceding the contraction.

He regards these effects as being in harmony with Hering's theory of dissimilation and assimilation, *i.e.*, that the relaxant effect of stimulation is a true inhibitory effect. In conclusion, he remarks: "The process of inhibition has its seat in the muscle itself. Biedermann's histological researches justify us up to a certain point in assuming that the stimuli of the two processes [D. and A.] are conducted through two kinds of nerve-fibres, and appear to confirm Gaskell's view that every tissue possesses

katabolic and anabolic nerves, of different structure and of different origin."

(8) **Boruttau** re-examines the point whether there is a physiological "lost time" in the motor end-plates (*Du Bois-Reymond's Archiv.*, 1892, p. 444,) and confirms the conclusion of Bernstein that there is. He ascribes the contradictory result of Hoisholt to the fact that the latter compared the effects of direct and indirect excitation of muscle with stimuli of insufficient strength. Piotrowski, experimenting on the adductor muscle of the crayfish claw, comes to a similar conclusion as to the existence of a physiological lost time.

(9) **Sherrington** (*Proc. R.S.*, Feb. 2 and April 15, 1893). With reference to the correlated action of antagonist muscles Sherrington's latest observations on monkeys are of considerable neurological interest.

He finds that the physiological state of the extensor muscles of the thigh (as tested by the knee-jerk) is influenced by the physiological state of the flexor muscles; thus paralysis of the flexors by nerve-section entails exaggerated response of the extensors, while that response is diminished or abolished during artificial contraction of the flexors, or by their passive extension, or by central excitation of their nerves; and he finds that by the proceeding last named the reflex tonus of the extensors is increased.

Still operating on the monkey he gives another and somewhat different illustration of the mechanism of functional correlation. After section of the 3rd and 4th cranial nerves of the left side, leaving therefore the 6th nerve and external rectus muscle alone active upon the left eyeball, he finds that appropriate cortical excitation will still produce conjugate deviation of the eyes to the right, viz., that presumably the left external rectus muscle was inhibited by the cortical excitation. This, it need hardly be said, is not offered as proof that the 6th nerve has an inhibitory or anti-motor action on the external rectus muscle; it is a physiological illustration of arrest of action by cerebral excitation, at a particular part of the muscular periphery experimentally picked out from the group of physiological movements in which it normally plays part. The experiment shows that in the lateral deviations of the eyes, the opposing muscles do not contract but give way, and it is left an open question whether the interference phenomenon is cortical or bulbar or muscular.

(10) **Charpentier** (*Archives de Physiologie*, 1892, p. 541), in prosecution of his optical investigations, publishes two very

beautiful and easily repeated experiments demonstrative of a slow oscillatory physiological process in the retino-cerebral apparatus.

(1) A disc $\frac{3}{4}$ black and $\frac{1}{4}$ white, revolving once in one to two seconds, viewed by direct sunlight, exhibits on the white quadrant a narrow black sector near the black edge that has just passed in front of the eye, and separated from that edge by a narrow white sector. This is presumably due to a rebound effect of the nature of an excitatory oscillation in the retino-cerebral apparatus; the first effect at the arrival of the white border is white, but this first effect is followed by an after-effect that is black, even *during* the continued white stimulus. Estimating from the rate of revolution and the apparent position and extent of the black band in the white quadrant, the positive and negative phases of the excitatory oscillation are found each to have a duration of 0.015 sec., indicating an oscillation frequency of 33 per cent. It is probably in consequence of an oscillation of this nature that a single bright stimulus causes a double or reduplicated bright sensation.

(2) A black disc, with narrow open sectors, revolving in front of the eyes, fixed upon a white surface illuminated by direct sunlight, gives rise to the sensation of a magnificent purple-violet field, when the rate of revolution is such that the retina is stimulated between 40 and 60 times per second, *i.e.*, when each stimulus occurs during the negative after effect of the preceding stimulus. At higher and at lower frequencies the field is colourless, and at the optimum frequency a white or yellowish space is seen in the centre of the purple field, corresponding with the yellow spot, in which the retinal purple is deficient. Charpentier is of opinion that the coloured sensation is due to entoptic vision of the retinal purple.

By calculation from another series of experiments, Charpentier finds that the excitatory oscillation, with a period of 0.028 sec. and a frequency of 36 per sec., is propagated from its seat of origin with a retinal velocity of 72 mm. per sec., and a wave-length of two mm.

(**11**) **R. Dubois,** *Anatomie et Physiologie de la Pholade Dactyle*, Paris, 1892.—This important monograph is full of observations on the dermatoptic properties of the integument of a well-known mollusk (Pholas Dactylus) that are of great importance to the general neurologist; the author develops at some length a new theory of retinal excitation, based upon his own observations, and reminding one of the hitherto isolated facts first pointed out

by Engelmann and van Genderen Stort with regard to the retractility of the retinal cones of the frog's retina. We cannot in a brief notice do justice to this memoir, but we may give the following summary of one of the author's main conclusions. There are very remarkable structural and functional analogies between the retina and the integument; an external layer of pigment-cells is connected with a superficial layer of muscular fibres that contract in response to luminous stimuli. This initial local contraction constitutes a mechanical stimulus to subjacent nerve-cells, and is followed by a secondary total contraction of the longitudinal musculature of the siphon. Histologically the dermic element excitable by light is composed of: (1) pigment-cell, (2) contractile fibre, and (3) nerve-cell. Functionally the peripheral stimulus is a dermic phosphene analogous with a tactile phenomenon, viz., a mechanical excitation of the nerve-cell, by a movement of the contractile fibre, in consequence of a photo-chemical change in the pigment-cell.

A.D.W.

A Study of the Artefacts of the Nervous Sysem. By Ira Van Gieson, M.D. (Reprinted from *New York Medical Journal*, September and October, 1892.)—Under this title Dr. Van Gieson gives a study of the "Topographical alteration of the grey and white matter of the spinal cord caused by autopsy bruises."

The word "artefact" has been ingeniously coined to avoid the use of the originally similar word, "artifice," which, owing to the common tendency of words to degenerate or take a less worthy meaning as language develops, would imply some disingenuousness on the part of the observer.

The author believes that "true heterotopia" of the grey matter may exist, but that it is extremely rare. He admits the existence of only six recorded cases, of which he gives a short *resumé*. In all these the displaced grey matter was apparently from the posterior horn and in the posterior columns. They were very minute and are presumed to be examples of true heterotopia because of the absence of any bruising of the surrounding white matter. We fail, however, to see in what respect these small displacements differ from the larger artificially produced alterations, except in degree. The grey matter must be supposed to be more diffluent than has hitherto been suspected, or the remarkable appearances produced artificially by Dr. Van Gieson would be impossible. We now come to the very interesting part of the work devoted to the description of the deformities produced by

bruising. A large number of drawings are given of sections from cords, first accidentally injured in removing, and secondly subjected after removal to every form of experimental injury. The effects produced are very remarkable, and the most prejudiced reader cannot fail to be struck by the close resemblance of the appearances to those in the recorded cases of so-called heterotopia. Even such effects as duplication of the cord have been produced artificially and figured by Dr. Van Gieson.

Then follows an analysis of the recorded cases of heterotopia, with reproductions of the plates, which Dr. Van Gieson condemns as artificial and not pathological at all.

The impression left on the mind of the reader is that there is no such thing as heterotopia, and the practical lesson to be learnt is, in removing a spinal cord never touch, bend, or twist it, and lift it only by its dura mater.

H. H. Tooth.

Die Circulation im Gehirn und ihre Störungen. By Dr. Richard Geigel. *Virchow's Archiv*, vol. cxix., p. 93.

Die Regulirung der Blutbewegung im Gehirn. By Dr. B. Lewy. *Virchow's Archiv*, vol. cxxii., p. 146.

"Experimentelle Beiträge zur Lehre von der Blut-Circulation in der Schädel und Ruckgratshöhle." By Professor Hubert Grashey. *J. F. Lehmann, München*, 1892.

Geigel begins by pointing out that Althann, of Dorpat, writing in 1871, demonstrated that the condition of fulness of the brain vessels was no measure of the good or bad blood supply of the nervous elements; but that the point of importance is the ease with which oxygen is brought and carbonic acid removed from these elements.

This depends on two things:—

1. The chemical constitution of the blood.

2. The quantity of blood that passes through the capillaries in a given time.

Geigel proposes to designate a satisfactory flow of arterial blood through the capillaries as eudiæmorrhysis, too little as adiæmorrhysis—true anæmia cerebri, and too much as hyperdiæmorrhysis—true hyperæmia cerebri.

He then states a proposition as follows:—

Let g = the velocity of the circulation of the blood in the capillaries of the brain. This is directly proportional to the

arterial pressure a, and inversely proportional to the resistance w. Therefore $g = \frac{a}{w}$

But w depends directly on the amount of the intra-cranial pressure = d, therefore $w = f(d)$ and $g = \frac{a}{f(d)}$

And Fick has shown that the intra-cranial pressure is equal to the intra-arterial pressure, less the resistance which the tension of the arterial walls oppose to it, therefore $d = a - s$ and $g = \frac{a}{f(a-s)}$

From this it follows: 1st, that if the contraction force of the arterial wall gets less; intra-cranial pressure $= d = a - s$ will get greater and g will get less, that is, the circulation will alter in the direction of adiæmorrhysis: in other words, dilatation of an artery causes anæmia, not hyperæmia.

2nd, if the tension of the arterial wall becomes greater $d = a - s$ will become less and g will get greater, that is, the circulation will alter in the direction of hyperdiæmorrhysis, so that spastic contraction of the arteries of the brain will increase the velocity of the bloodflow through its capillaries.

Again, suppose the heart acts more powerfully, raising intra-arterial pressure, while at the same time the arterial walls increase their contraction, intra-cranial pressure will be the same, or greater or less, according as the arterial contraction equals, or is less or greater than the rise of intra-arterial pressure.

Hence, with increased contraction of arterial walls intra-cranial pressure decreases and capillaries widen. On the other hand, with diminished contraction of arterial walls intra-cranial pressure is raised, the capillaries are compressed, and the amount of blood circulating through them is diminished.

The writer points out that the symptoms of cerebral pressure are really due to the interference with the circulation which the pressure produces, and are not the direct result of the pressure on the cerebral substance: for the brain substance will bear the pressure of two atmospheres and more without harm, provided its circulation is not thereby interfered with.

The supply of oxygen and removal of carbonic acid are the points of importance, and these are equally interfered with by high intra-arterial pressure and by anæmia; hence the symptoms of these two conditions may much resemble each other.

He applies the same reasoning to the effects of embolism or rupture of a vessel.

By an ingenious arrangement of tubes, partly glass and partly elastic and expansile, passing through a closed container full of water, he shows that when one tube is blocked to represent an embolus the flow is contemporaneously diminished through the

other tubes which are not blocked, owing to the general fall of pressure in the closed container produced by the contraction of the vessel that is blocked.

Thus embolism of one intra-cranial artery produces a temporary diminution of circulation in all the other intra-cranial vessels, and thus "adiæmorrhysis" is the cause of the apoplectic shock of embolism.

The rupture of a vessel produces a similar general adiæmorrhysis by raising the intra-cranial pressure, for in normal conditions $d = a - s$, but when a vessel is ruptured s is more or less completely removed, and $d = a$, and the resulting adiæmorrhysis is the cause of the apoplectic shock, and this will be more severe the more rapidly d is made = to a.

He argues also that the same laws apply to the cranium of a child with the fontanelles open; that spastic contraction of arteries causes hyper-diæmorrhysis and paralytic dilatation adiæmorrhysis because the dura mater keeps up the internal pressure and acts very much as the closed skull in the adult.

Lewy takes a somewhat different view of the matter, for though he acknowledges that an intra-cranial artery cannot expand without taking space from other vessels, he points out that the space thus taken is so small that in physiological conditions it affects the cerebro-spinal fluid, but not the capillary circulation at all.

Again: when an artery enlarges, the blood meets with less resistance in passing through it; and it is possible that the lessened resistance in the artery more than compensates for any slight increase of resistance in the capillaries due to the expansion of the artery, and he points out that Geigel has overlooked this.

Conversely, contraction of an artery may increase the resistance more than the corresponding relaxation of capillaries diminishes it.

When the arteries all enlarge together, the lymph gets out of way and the capillaries are so numerous that they will bear a large amount of compression before they are so far closed as to hinder the circulation; and under these conditions narrowing an artery diminishes the blood stream and widening an artery increases it, so that arterial hyperæmia is possible. But beyond a certain point this does not hold, for if we imagine the arteries to enlarge so much that the veins are pressed flat absolute stasis will result: if the arteries now begin to contract, passage of blood will begin again and increase, and thus a narrowing of the arteries produces hyperæmia.

If, however, part of the intra-cranial space is taken up by a tumour, or again, if a large number of capillaries have been destroyed by injury or inflammation, then a smaller amount of arterial enlargement may seriously interfere with the capillary circulation.

Lewy thus acknowledges that Geigel's argument may hold for certain pathological conditions, but denies that it does so for physiological conditions.

He further points out that when the skull is open the brain is seen to undergo an increase of volume corresponding to each systole; but when the skull is closed there is no increase of volume, but the pulsatory quickening of the blood stream is carried on and becomes visible in the veins.

Lewy, as we have seen, considers that the arteries may possibly enlarge so much as to compress the veins and produce stasis; but Grashey shows that this result may ensue, not so much from dilatation of the arteries, as from rise of intra-arterial pressure.

When this pressure rises beyond a certain height the central veins begin to vibrate, and then the amount of blood streaming through is decidedly reduced. At this point it is possible that the symptoms of pathological brain pressure begin.

At this point dilatation of an artery will still further increase the pressure on the veins and do harm, so that here, to some extent, Grashey agrees with Geigel.

In other points he disagrees with Geigel, for he points out that hyper-diæmorrhysis is not proportional to arterial contraction, for if the spastic contraction of arteries is very great, the blood stream may obviously be stopped altogether: again, contraction or dilatation of arteries must obviously influence intra-arterial pressure, but Geigel argues as if this always remained the same.

Contraction of an artery diminishes the pressure in it, dilatation of one increases the pressure in it; for this reason dilatation of an intra-cranial artery increases the pressure on the veins and does harm, while the local application of cold may do good by contracting the vessels.

According to Grashey, the quantity of blood that streams through the cerebrum in a given time rises with the pressure in the aorta up to a certain point: but when the pressure rises still further the central veins (at or near their point of entry into the sinuses) begin to vibrate, and the amount of blood passing through them is greatly diminished. The vibration appears to be due to the alternate contraction or dilatation of these veins according as the pressure inside them overcomes, or fails to overcome, the compressing force outside them.

Under these conditions the veins behind the point of compression, and the capillaries behind them, are overfilled with blood, which however is able to move but slowly, and the conditions are thus unfavourable for the nutrition of the brain.

Grashey further believes that the stasis thus brought about may cause an increase in the quantity of the cerebro-spinal fluid, and this will still further add to intra-cranial pressure, so that things tend to get worse and worse once this vicious circle is established by the pressure of high arterial tension; and mere removal of high arterial pressure may not now suffice to remove all the symptoms of intra-cranial pressure.

What is said above of the effects of dilatation of a cerebral artery is obviously true of a local dilatation only; for when the arteries all over the body dilate a fall of blood pressure results and the pressure on the intra-cranial veins is diminished.

All these writers agree that the quantity of blood in the brain is of little consequence compared with the freedom of its circulation, and the ease with which it can give out oxygen and take up carbonic acid. And two of them agree in believing that high intra-cranial pressure may compress the veins and produce stasis and hyperæmia in the smaller veins and capillaries behind them, and that such a condition is unfavourable for the nutrition of the brain even though its vessels now contain an unusually large amount of blood.

A. Haig.

Contribution a l'étude Anatomo-pathologique et Clinique des différentes Variétés de Cécité Verbale. par M. J. Dejerine (*Mémoires de la Soc. de Biologie, Séance du 27 Février*, 1892).

This is a most interesting and instructive case of word-blindness. An elderly man, intelligent and well educated, suddenly became unable to read. This brought him under the notice of M. Landolt, who later, sent him to M. Dejerine. He displayed no noteworthy error of refraction, and his direct vision was fairly acute, but he had "*une hémiopie*[1] *homonyme latérale droite*,

[1] The left halves of both retinæ were affected. It seems desirable here to allude to the extremely unsatisfactory state of our nomenclature of symptoms of this class. With some writers hemianopia and hemiopia are to be interpreted with reference to the retinæ. Thus, for example, *left* hemianopia would signify paralysis of the left halves of both retinæ. With others the same terms are to be interpreted with reference to the field of vision, and *left* hemianopia would then imply darkness of the *left* half of the visual field, and so would connote paralysis of the *right* halves of the retinæ. Those who employ hemianopia in the former of the two senses not infrequently use hemianopsia, to be interpreted as referring to the field of vision. But even were this distinction between left hemianopia and left hemianopsia uniformly observed, there yet remains an objection to the use of the words, namely, that there is no etymological distinction between them to warrant their employment to connote opposite conditions of the retinæ. Analogy with myopia and kindred words may appear a justification for hemianopia, denoting half-retinal blindness; but the ambiguity arising, both from its variable significance, and from its likeness—in sound, as well as in appearance—to hemianopsia, is a real, and not merely an æsthetic, difficulty attending its use. Now amaurosis is already in vogue as a term restricted to a retinal condition,

qui n'est pas absolue," and which was accompanied by a co-extensive, and absolute achromatopsia. Further examination revealed alexia, not merely of words, but of letters and musical signs as well, with the last of which he had previously been unusually familiar. Yet he could recognise and name without delay or error all other familiar objects, drawings not excepted. He could write, both spontaneously and from dictation, almost as well as before his trouble, but was totally unable to read what he had written. Copying, since he could not read, was next to impossible; but he succeeded by literally *sketching* each letter to transcribe slowly, and somewhat illegibly. Cyphers, however, he could read, write, and copy with comparative ease; and he was able to perform easy arithmetical operations with accuracy, though with extreme slowness. His hearing was acute, and in every respect normal; his speech in no way defective; and his powers of *mimique* unimpaired. He had neither hemiplegia, nor ataxy, nor were there any sensory symptoms beyond the visual troubles above described. These symptoms persisted without marked alteration for four years, during which he was under constant observation. Ten days before death, he was suddenly seized with total agraphia, and paraphasia, without any word-deafness, and without any loss of his faculty of *mimique*. There also appeared, transitorily, motor and sensory troubles in the right limbs, and also a complete *Seelenblindheit*, which, too, disappeared before death. The patient sank finally into coma, which passed gradually into death.

At the autopsy, two lesions were discovered in the left hemisphere; the one, of long standing, involved the extreme hind end of the hemisphere; the other, a recent one, being situated more anteriorly. The former lesion occupied, externally, the posterior extremities of the first and second occipital convolutions and the sulcus between them; intero-inferiorly, it involved the base of the cuneate lobule, the walls and floor of the calcarine fissure, and the posterior end of the occipito-temporal gyri, leaving however the limbic lobe intact. The optic radiations were much degenerated, and atrophied, as was also a portion of the splenium of the corpus callosum. The recent lesion implicated the supramarginal, and angular gyri, and the second annectant gyrus. It extended inwards as far as the ependyma ventriculorum. The rest of the brain was normal.

The account is well illustrated by wood-cuts, and to it the reader is referred for fuller details. The association of the old lesion with hemi-amblyopia and cæcitas verbalis *sine* agraphiâ, is most interesting, and it is believed that the author's case is the

and hemi-amaurosis, besides being unambiguous, is not objectionable from a classical standpoint. Scotosis, too, from its sisterhood to scotoma, a term confined to a condition of the visual field, is familiar to all; and hemi-scotosis is as safe from misapprehension as is hemiamaurosis. It would be better, therefore, both from the utilitarian and from the æsthetic point of view, to designate such a symptom *left* hemi-amaurosis, or *right* hemi-scotosis, expressions both of which connote blindness of the *left* sides of the retinæ. There is no objection, however, to the employment of the convenient adjective *hemiopic* provided that the paralysis be accurately defined by less ambiguous terms.

first such, in which an autopsy has been obtained, and in which therefore the lesion has been described. It cannot, however, be regarded as a strictly unilateral lesion, for though the right hemisphere is described as "*intact à la surface, et sur les coupes*," a degeneration of the splenium is noted, which presumably extended into the right optic radiations, and therefore constituted a sub-cortical lesion in the right hemisphere. The connection between the recent lesion and cæcitas verbalis *cum* agraphiâ is accentuated by references to like lesions, associated with similar symptoms, recorded by M. Dejerine himself, Berkhan, and Serieux. But the *necessary* association of "*angular*" lesions with these symptoms is most difficult of proof. For example, in the case in point, the softening reached inwards as far as the ventricle, and so was, in effect, not merely a parietal lesion, but a sub-cortical occipital lesion as well, involving as it must have done many of the optic fibres in their path from the internal capsule to the occipital cortex. The history, too, of brain-physiology, reminds us of the tendency to ascribe to the gyrus angularis, an importance possessed to a greater extent by the regions posterior to it. And consideration of the anatomical relations of this portion of the encephalon, further discourages belief in the exclusive part played by the angular lesion in such cases. For, any lesion that destroys, to its full depth, the convolution in question, lies alarmingly near the fibres of the optic radiations, which at this level have not diverged to any great extent, and which consequently would be injured in large numbers, by a very slight extension of the lesion inwards. Moreover, and this especially in the central nervous system, the apparent, molar, anatomical confines of a given lesion, are by no means its true, molecular, physiological limits. Bearing in mind then, the difficulty of assuring ourselves that a lesion apparently limited to the angular convolution, is really confined to that gyrus, and keeping in view the direction taken by erroneous notions in the past, it seems more reasonable to recognise that simple alexia and hemi-amblyopia, and the same symptoms plus agraphia and paraphasia, may result respectively from less extensive and more extensive posterior lesions, than to assume that they correspond to "*deux lésions anatomiquement distinctes.*" It would be interesting to learn whether the final symptoms included any aggravation of the hemi-amblyopia.

In conclusion, we would express our real appreciation of M. Dejerine's valuable contribution to brain-literature.

WILLIAM LEGGE SYMES.

We are glad to announce that the *Report of the Proceedings of the Congress of Experimental Psychology* held in London in August last, is being published by Messrs. Williams & Norgate, of Henrietta St., Covent Garden. The price to others than members will be five shillings.